





Samuel Graham ✓









FIG. 1.



Case I. Photograph showing the conjunctival ecchymoses and peculiar discoloration of face, lips, and neck, which follow forcible compression of the chest with inhibition for several minutes. Page 10.

# INTERNATIONAL CLINICS

## A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND  
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-  
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PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
OTOLOGY, RHINOLOGY, LARYNGOLOGY,  
HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

**BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD**

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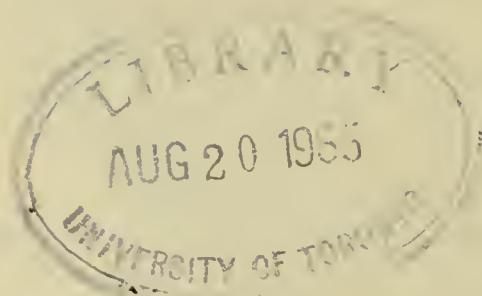
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# Clinics

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## A CLINICAL STUDY DURING SIX YEARS OF A PATIENT WITH AURICULAR FLUTTER PASSING INTO FIBRILLATION, AND POST- MORTEM FINDINGS

BY DR. JAMES E. TALLEY

Clinic at the Presbyterian Hospital

---

HAVING finished the routine discussion of auricular flutter and its treatment, we will devote the rest of our session to a consideration of the clinical course, method of termination, and the post-mortem findings in a case of auricular flutter which passed into fibrillation and persisted for six years. How long the flutter had existed before the patient reported, I have no way of knowing, but have a note on having seen the patient once in February, 1909, when he gave a history of attacks of palpitation followed by dyspnœa, which recurred every few days. The pulse was from 64 to 70 and irregular. The irregularity was probably due to premature beats. The apex was in the normal position, there were no murmurs, his systolic pressure was 180, but this was a single observation. His liver was normal in size, his nervous system normal, his sleep good, and digestion fair. Urine was acid, 1.010, showed a faint trace of albumin and a few hyaline casts.

The patient, a man now of 75 years, an Englishman by birth, and a weaver by trade, reported again April 18, 1915, for distress in the chest and dyspnœa, which had persisted for some time and had grown worse, and for which he had had no systematic treatment. The apex rate at this time was from 144 to 184. The apex beat was in the fifth interspace, about four inches from the mid-sternal line. His arteries were stiff. The pulsations in the over-distended veins of the neck were rapid. There was no œdema, and the lungs were normal.

He had had an attack of acute auricular rheumatism at the age of fourteen, and again at the age of twenty-six. The left hand and left foot still showed some prominence of the joints. In the

attack when twenty-six years of age, the legs were swollen, and he was in a hospital in Birmingham, England, for at least five weeks. There was no history of venereal disease. Several examinations of the urine during the next six years showed a specific gravity of 1.010 to 1.015, a mere trace of albumin, with a few hyaline casts.

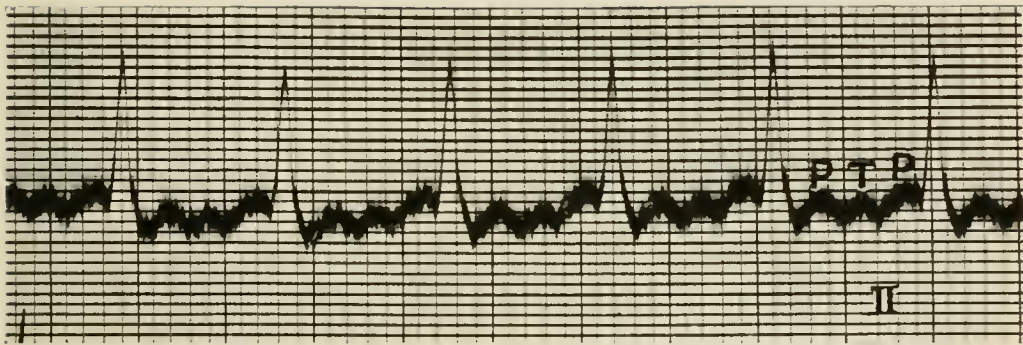
I had supposed that the patient had been admitted to the house for examination, but was mistaken in this, so that no reports on the Wassermann, blood ureanitrogen, or phenolsulphonphthalein were found. His average blood-pressure was low; systolic, 120; diastolic, 90.

The first electrocardiogram (Fig. 1) was taken April 19, 1915, and shows auricular flutter with 2:1 block. He was dyspnoëic, complained of pain in the chest, and exhaustion on the slightest exertion, but preferred to return home. He was already taking digitalis, which he had begun on the seventeenth, and this was continued so that he had taken 4 drachms of the tincture when the electrocardiogram of April 22, 1915, was taken. His ventricular rate was now 120 and irregular. This electrocardiogram showed auricular flutter with 3:1 and 4:1 heart block. The same as Fig. 2. The same dosage of digitalis was continued, and three days later, April 25, 1915, an electrocardiogram showed the mechanism had again changed to auricular flutter with 2:1 block, with the original ventricular rate of 165 and auricular, 330. Same as Fig. 1. Fig. 2, April 27, 1915, shows that the mechanism had changed back to auricular flutter with 3:1 and 4:1 block and premature beats.

An electrocardiogram taken April 30, 1915, showed auricular fibrillation as in Fig. 3, so that the digitalis was now withdrawn, but a regular rhythm never returned and the fibrillation persisted until his death, practically just six years later. Fig. 3 was taken May 20, 1915, after the patient had had recourse to digitalis on his own volition.

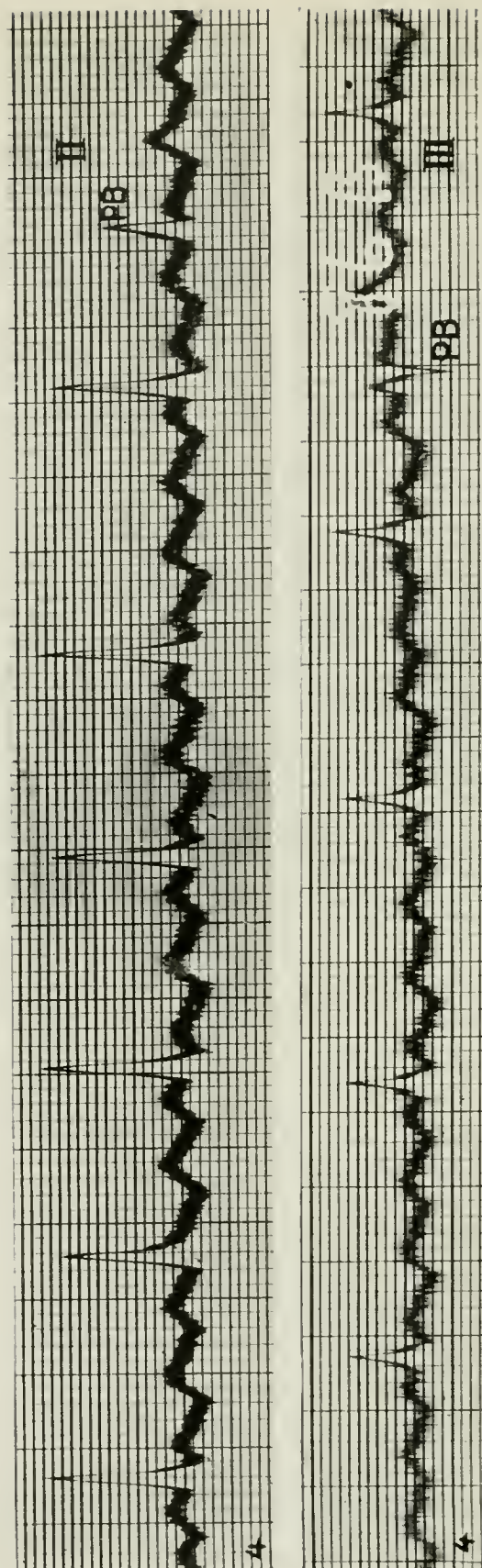
During the year 1916 there were no curves taken, but he continued to fibrillate, as he was seen two or three times during the year. On account of my absence during a part of 1917 and all of 1918, no curves were taken, but we have curves for 1919 and 1920 (Fig. 4). He was seen a number of times during these two latter years and each examination showed auricular fibrillation. Many curves

FIG. 1.



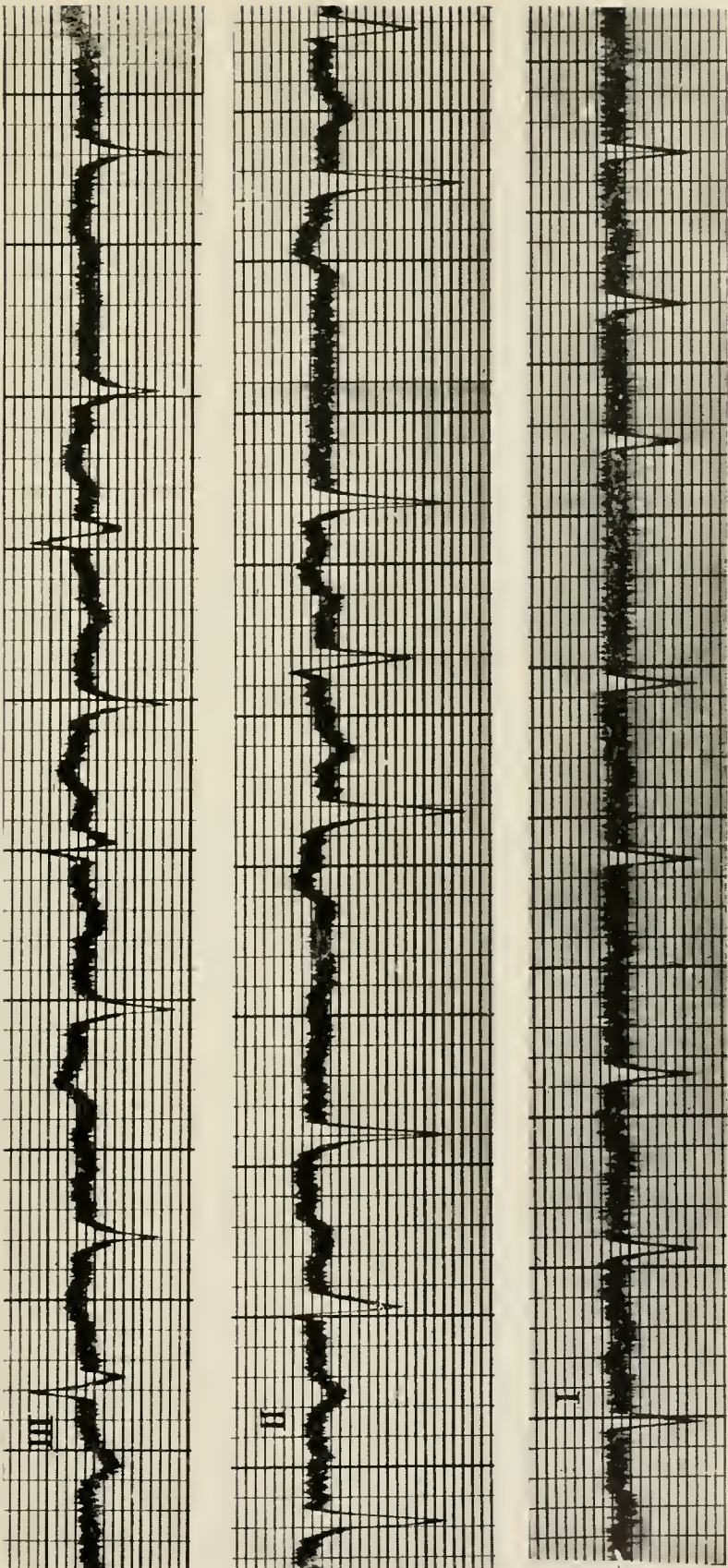
Apr. 19, 1915, Lead II. Ventricular rate 1:65; auricular rate, 330; 2:1 heart block.

FIG. 2.



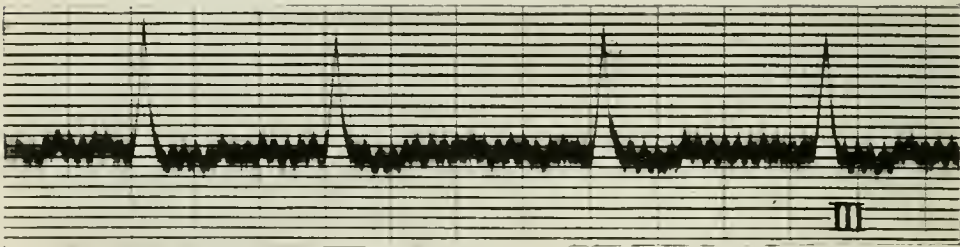
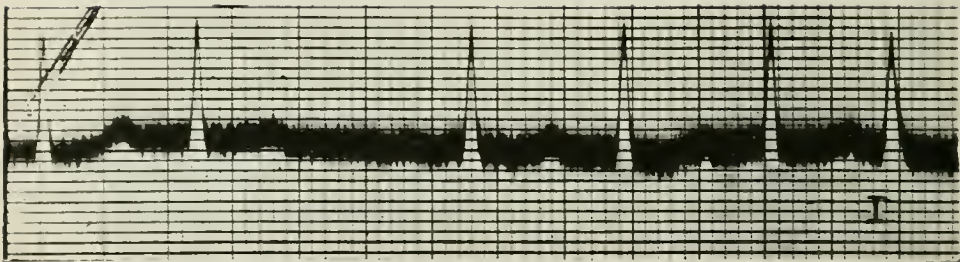
Apr. 27, 1915, Leads II and III. Auricular rate, 330; ventricular rate, 80 to 88. Degree of heart block, 3:1, 4:1. PB. are premature beats.

FIG. 3.



May 20, 1915, Leads I, II and III. Ventricular rate, 100, irregular. Auricular fibrillation. Coupling.

FIG. 4.



May 19, 1920, Leads I and III. Ventricular rate, 80, irregular, Auricular fibrillation.

taken during these years showed that the character of the fibrillation remained the same. In February, 1917, after the emotional excitement of reading a political speech, the patient was seized with motor aphasia, with weakness in his left arm, and sluggish knee jerks, but the left soon became slightly exaggerated. At this time his apex beat ran from 150 to 160, but on the same day it fell again to 120 to 130. It was apparently fibrillation throughout. Recently he had complained of more marked dyspnoea and easy exhaustion.

One suspects that this attack was due to temporary arterial spasm inasmuch as the patient had entirely recovered in a few days. The average apex rate throughout the six years whenever taken was from 110 to 120 and irregular. He had an occasional exacerbation when the pulse would be much higher. His curves also showed some premature beats, but these were not numerous. Furthermore, his pulse deficit was never high until 1921. It kept below 20 until his last illness, when there was a tremendous jump in both rate and pulse deficit.

Throughout the six years the patient's physical activities were circumscribed to going up and down one flight of stairs and to walking a few blocks. His mentality was unimpaired. He was an assiduous reader, and he put in part of his time constructing simple mechanical devices while seated.

Throughout the six years he took digitalis according to his own subjective symptoms, for longer or shorter periods. An increase in his pulse rate, dyspnoea, sub-sternal distress, or slight puffiness of the ankles were his indications. On April 21, 1921, while apparently in his usual health, he had an apoplectic attack with aphasia, resulting in a completely paralyzed left arm and a left leg almost completely paralyzed. His apex rate three days before had run up to 140, and his radial rate was 112, making a pulse deficit of 28. On the day of the attack, the apex rate was 152 and the radial had fallen to 88, making a pulse deficit of 64. This rapid irregular rate continued, and the patient succumbed on April 24, 1921, at the age of 81 years and one month, after almost exactly six years of fibrillation.

As we have already seen, when digitalis is given to a case of flutter there is first ventricular slowing by increasing the degree of

block, while the original auricular rate persists. Frequently, if digitalis is still continued, the auricular flutter will pass into auricular fibrillation, and often if the drug is now withdrawn, the normal rhythm returns. In this case it did not. Flutter may pass into fibrillation without the patient having had digitalis. In experiment, auricular flutter may be converted into auricular fibrillation by stimulation of the vagus and occasionally the reverse mechanism has been seen. The transitional curves from flutter to fibrillation are infrequent in human curves. This relationship between auricular flutter and auricular fibrillation is more easily understood since the acceptance of the idea of "circus movement" of the excitation wave in the auricle.

Lewis,<sup>1</sup> having satisfied himself that experimental flutter of the auricle consists of a "circus movement" of the excitation wave around some natural ring of muscle in that chamber, has gone further, and by a careful analysis of curves from patients concludes that the "circus movement" in human flutter is usually around the two cavæ and that probably this movement is usually, though not necessarily always, down the *tænia terminalis* and up the left auricle. By further experiments and careful analysis of human curves he concludes that auricular fibrillation is also due to a "circus movement," but this circulating wave does not run the constant course in auricular fibrillation that it does in auricular flutter.

For the autopsy and the careful study of the organs in this case, and the following pathological report, we are indebted to Dr. John Eiman, Director of the Pathological Department of this hospital.

There is only one suggestive thing about the pathological findings and that is that the degenerative changes were extreme in the right auricle.

Another point of interest was the extreme degree of fibrosis and arterial degeneration.

The autopsy was performed 54 hours after death on an embalmed body.

*External Appearances.*—White man, about 60 years old. No œdema, jaundice, or cyanosis, weight about 125 pounds. Eyes: Pupils equal, 5 mm. in diameter, slight arcus senilis.

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<sup>1</sup> LEWIS: *Heart*, vol. vii, No. 4.

*Internal Appearance.*—Abdomen flat, wall thin; peritoneum pale, smooth, glistening, some fibrous adhesions around gall-bladder and spleen. The liver is 1.5 cm. above the costal margin in the mid-clavicular line. Height of diaphragm right side, fourth rib; left side, fifth rib.

*Thorax.*—The antero-posterior diameter slightly increased and the costal angle somewhat obtuse.

*Pleuræ* normal. *Pericardium* is entirely covered by the lungs causing entire obliteration of area of absolute dullness. Pericardium is pale, smooth, glistening and contains no fluid. It extends 4.5 cm. to the right of the mid-line.

*Heart.*—Shape is somewhat distorted due to embalming. Measures 15.2 x 10 x 7.3, weight 372.5 gms. *Epicardium.*—Pale, smooth, glistening. Shows fairly large irregular areas of thickening. There are excessive amounts of subepicardial fat tissues. *Coronary arteries* show advanced arteriosclerosis. *Orifices.*—Mitral, 10; pulmonary, 7.8; tricuspid, 12.5; aortic, 7.5. *Mitral leaflets* show diffuse thickening throughout, but especially along the bases. *Right anterior pulmonary leaflet* shows thickening and induration along the edges. *Tricuspid leaflets* show no noteworthy lesions. Thickness of the left ventricle varies from 8 mm. at the tip to 23 mm. Thickness of the *right ventricle* is 7 mm. Auricles are about 1 mm. thick. The *cordæ tendinæ* on the left side are shortened and thickened. The *palpillary muscles* show marked fibrosis. *Myocardium* is rather pale and hard (partly due to embalming). The *endocardium* shows slight thickening over the septum below the aortic leaflets.

*Lungs.*—Emphysematous. *Stomach* and *intestines* appear normal. *Liver* is diminished in size, nutmeg in appearance, firm surface is finely and uniformly granular.

*Left Kidney.*—10 x 5.5 x 4.2 cm. It is markedly deformed due to deep scars. Capsule strips with difficulty and leaves deep scarred surfaces. The kidney substance between the scars shows fine granulations. Section, pale. Thickness of cortex 2 to 3 mm. Thickness of medulla is 15 mm. There is a small infarct at the bottom of a deep scar. Renal vessels stand out like pipe stems. Excessive amounts of fat in the pelvis. Ureter shows no gross lesions.

*Right Kidney.*—Like its fellow.

*Spleen*.—10 x 5.7 x 4 cm. "Zuckerguss" covers the entire outer surface. At the thickest point capsule measures 6 mm. Section surface purplish. Follicles not visible.

*Pancreas*.—Fibrosis.

*Aorta* shows advanced arteriosclerosis with calcareous plaques in the abdominal aorta.

*Microscopic*.—*Heart*.—Epicardium shows localized patches of thickening due to overgrowth of dense, hyalinized connective-tissue. There are excessive amounts of subepicardial fat but there are not any evidences of fatty infiltration of the myocardium.

*Coronary arteries* show very marked thickening of the intima due to overgrowth of connective-tissue. In areas showing greatest thickening there are seen deposits of fatty material and calcium salts. The medium shows similar changes in many places. Due to irregularities of the wall the shape of the lumen is markedly distorted. *Myocardium*.—*Left ventricle* shows more or less diffuse increase of interstitial connective-tissue which in many places is very dense and assumes the character of small scars. The arterioles show marked sclerotic changes. The muscle cells vary in size, some are larger than normal, but most of them are considerably smaller. Lipochrome pigment is seen only in moderate amounts. Around microscopic scars there is seen a feeble attempt of regeneration of muscle fibres. Small areas show fragmentation. The *papillary muscles* of the left ventricle especially show much more marked fibrosis than the walls. The *right ventricle* shows changes similar to those of the left, only not so marked. The *auricles*, especially the right, show very marked diffuse fibrosis. All remaining muscle cells are either atrophic or distorted (Fig. 5). *Endocardium* is moderately and irregularly thickened due to overgrowth of dense connective-tissue.

*Histological Diagnosis*.—*Heart*.—Coronary sclerosis. Chronic interstitial myocarditis most marked in right auricle. Atrophy of muscle fibres. Chronic patchy pericarditis. Chronic endocarditis. Fragmentation of myocardium.

*Lungs*.—Emphysema.

*Liver*.—Chronic passive congestion.

*Spleen*.—Chronic perisplenitis, chronic interstitial splenitis.

*Kidneys*.—Chronic diffuse (arteriosclerotic) nephritis.

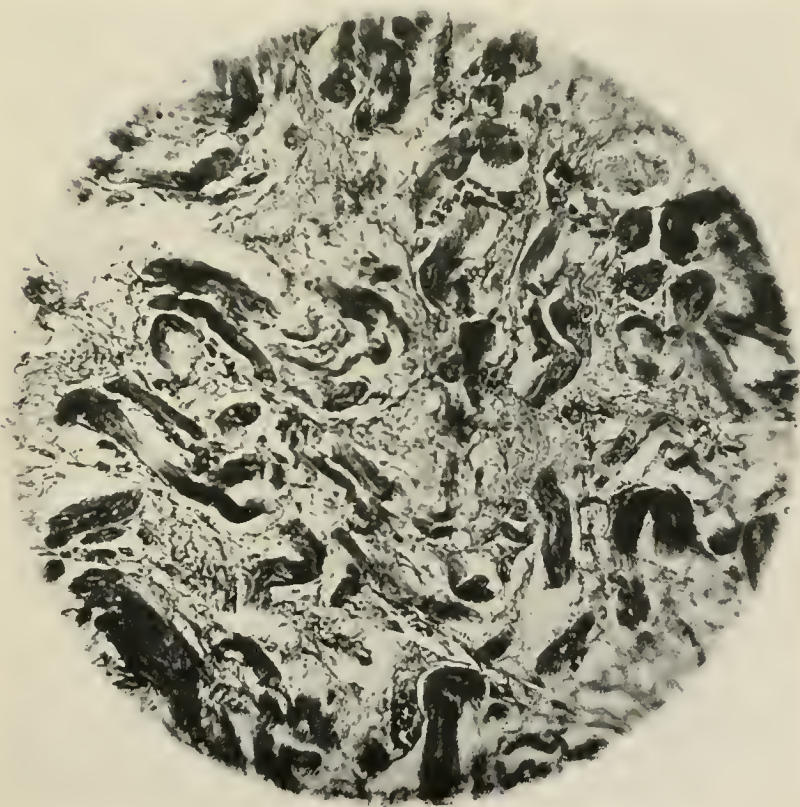
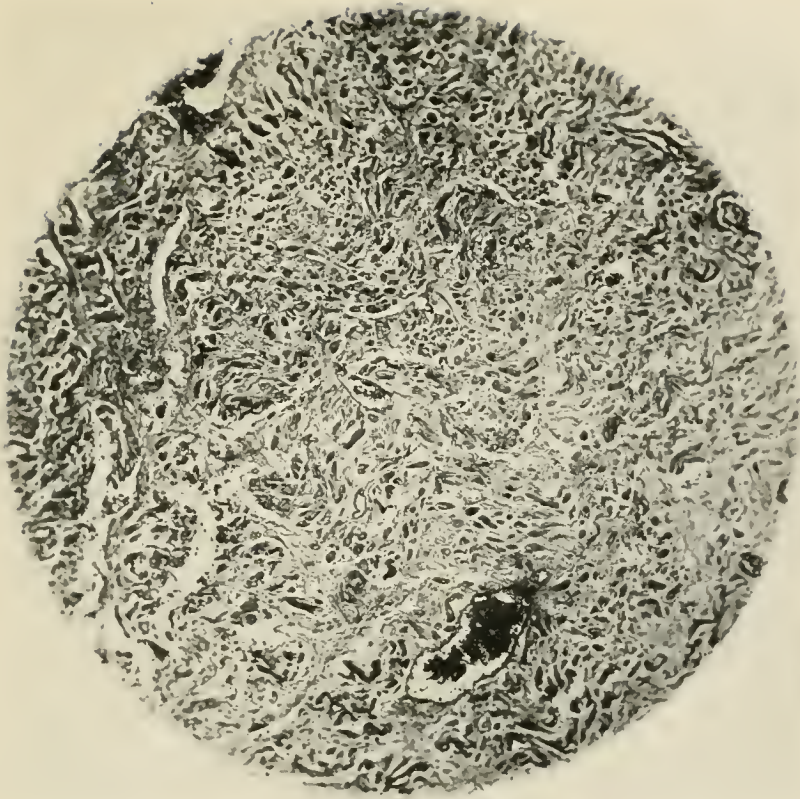


FIG. 5.

Low and high power of right auricle showing marked fibros.



## THE TREATMENT OF UMBILICAL HERNIA

BY LEIGH F. WATSON, M.D.

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ON account of the favorable location of umbilical hernia it was more frequently subjected to operation in ancient times than the other varieties. Celsus used the elastic ligature in the treating of umbilical hernia. He first placed the patient on his back to favor the return of the omentum and intestine into the abdominal cavity. Usually he opened the sac to make certain the contents were reduced. Then he applied a double ligature to the sac and seared the stump with caustics or the actual cautery to secure a firm cicatrix.

This method was widely used, and kept alive through the centuries by the writings of Paul of Ægina, a celebrated Greek surgeon who practiced in Alexandria during the seventh century; Avicenna, who lived in the ninth century, the most famous of the Arabian physicians; Albucasis; Guy de Chauliac, the eminent French surgeon who wrote a treatise on surgery in the twelfth century; Pare, the father of French surgery who lived in the fourteenth century; Garengeot; Saviard and Petit. Segond has fully described the ancient forms of treatment which were gradually displaced by other methods and were only revived with the advent of the antiseptic period of surgery.

Infantile hernias may vary in size from the "umbilical button," which is no larger than the tip of the finger, to a tumor as large as a fetal head. Small hernias are usually spherical in shape, becoming conical as they increase in size. The sac is almost always adherent at the fundus or at the cicatrix.

In small infantile hernias the sac is empty except when a knuckle of intestine is forced into it as the child cries or strains. Larger infantile hernias may contain one or more loops of small intestine and rarely a portion of transverse colon. If the hernia is neglected and allowed to increase in size it may have the same contents as the adult variety.

Infantile umbilical hernia is usually cured spontaneously before

the third year. Very rarely does it persist in adult life. As strangulation is rare, the prognosis in these cases is good, if the child is properly fitted with a binder or support.

To diminish the chance of the development of hernia, care should be exercised to avoid traction on the cord; it should be ligated one or two inches from the abdomen, under strict aseptic precautions, and protected with sterile dressings until it separates and the granulating wound heals. An antiseptic dusting powder is often useful. The dressings can be kept free from urinary contamination by covering the lower portion of them with collodion, oil silk, waxed paper or adhesive tape. A snug abdominal binder should be applied and allowed to remain until the umbilical cicatrix is firm. As oftentimes umbilical hernia does not appear for weeks or months after birth, I have found that the binder should be left on some months longer than has been the custom until the child is at least six months old.

The mechanical treatment of small infantile umbilical hernias results in a high percentage of cures, if it is used early. A ring with a sharp distinct outline usually closes more slowly than one that is indefinite and irregular. De Garmo states that the length of time required depends largely on the age of the child. If treatment is begun at the age of three months, about three months' time is required for a cure; if the child is six months old, the truss will have to be worn for at least six months; if treatment is not begun until the child walks, it will take a year's time, or more, to effect a cure.

The simplest treatment in children under a year old, and probably the best, is to reduce the hernia and apply firm adhesive straps, which should reach from the posterior axillary on one side, to the same point on the opposite side, extending at least one inch above and one inch below the edges of the umbilical ring. The straps should not extend entirely around the body because of the danger of an increase in intra-abdominal tension, which might cause inguinal hernia.

Many of the older writers, especially the Germans, advocate the folding in of the skin and subcutaneous tissues in the form of a pad, before applying the adhesive straps. The disadvantage of this method is that the infolded skin becomes irritated and is a

source of pain and discomfort to the little patient. The best plan is to make a pad or compress, which must always be larger than the hernial opening; this pad can be made from a large flat button or a hard rubber or cork disk, and covered with some soft material to prevent chafing. If it is smaller than the hernial opening, it will tend to force the ring more widely open, and defeat the purpose for which it is intended. With the hernia reduced, the compress is placed over the centre of the umbilicus and held in position by a disk of adhesive plaster which is at least one-half inch larger than the pad. The long adhesive straps are applied as the compress is pressed inward and the skin of the abdomen drawn in from each side. If the child is restless and crying, it is hard to adjust the straps while he is lying down, but it is a simple matter if he is suspended by his feet. In this position the hernia does not protrude, and the child does not cry or kick and intra-abdominal pressure is not increased.

Some physicians use an elastic belt with an air or water pad, which fits over the hernia. After the child is one or two years old, a truss is usually more satisfactory than the adhesive strapping.

A bandage, belt or truss must be changed frequently to suit the growth of the child. In children under a year old, whatever appliance is used should be kept on day and night; older children should wear a truss in the day time, and a light belt at night. The skin must always be kept clean and dry. If the hernia is stationary in size, or growing larger, after the child is five years old, operative treatment is indicated. After this age cures by a truss are very rare, and when they are affected, there is more chance of recurrence than in those patients who were cured during the first year or two of life. It is not unusual to see adults with recurrent umbilical hernia that was apparently cured in childhood by truss treatment.

Operative treatment of infantile umbilical hernia is indicated when the hernia is large, and causes frequent attacks of colic, vomiting and symptoms of partial intestinal obstruction; when the hernia cannot be held satisfactorily by a truss or bandage, and when there is little prospect of cure by mechanical measures.

After the age of five, the likelihood of cure by non-operative means is very slight, and unless the hernia is very small, and diminishing quite perceptibly under treatment, operation should be resorted to.

# TRAUMATIC ASPHYXIA, RUPTURE OF LUNG, DISLOCATION OF CLAVICLE; PYONEPHROSIS, URETERAL STONE; HYPOSPADIAS

BY CHARLES DAVISON, A.M., M.D.

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## TRAUMATIC ASPHYXIA, RUPTURE OF LUNG, DISLOCATION OF CLAVICLE

THIS patient is a boy twelve years of age, who sustained severe injuries in an automobile accident. He was playing ball in the street and ran, head on, into the radiator of a moving automobile. He was picked up and brought into the hospital at once. An immediate physical examination developed the following conditions:

1. Shock. The child was almost in extremis. He was gasping for breath. His respirations were weak, shallow and rapid. His pulse was irregular and almost imperceptible. His skin was bathed with cold, clammy perspiration.

2. Dyspnœa. His efforts at breathing were frantic and irregular. Decubitus was impossible. He had to be kept constantly in the sitting position or he would stop breathing. The frequent use of oxygen apparently prevented immediate death.

3. Cyanosis. A marked bluish discoloration of the skin, limited to the scalp, face, lips and neck was present. It disappeared in four or five days (Frontispiece).

4. Conjunctival ecchymoses. Both eyes showed marked ecchymoses (Frontispiece).

5. Expectoration. There was a marked expectoration of frothy, blood-stained sputa. There was a constant, distressing, hacking cough.

6. Cellular emphysema was present over the neck and chest. It became limited in a few hours and disappeared in four or five days.

7. Collapse of lung. Examination of the right side of the chest by inspection, percussion and auscultation indicated collapse of the right lung. There seemed to be no movement of the right chest wall

during attempts at respiration. Percussion gave a tympanitic sound over the right side, except at the lowest part, which showed a limited dull area. There were no respiratory sounds to be heard with the stethoscope on that side of the chest. Examination of the left side of the chest indicated partial collapse of the left lung. There was very slight movement of the left chest wall during attempts at respiration. The upper part of the chest was tympanitic but the lower part was dull on percussion. Auscultation developed a few coarse râles in the bronchial tubes but no vesicular sounds were audible.

8. Dislocation forward of the sternal end of the left clavicle.

9. Bruises about the head and chest. There was no apparent injury to the abdomen or other parts of the body.

The boy was propped in the sitting position with ice packed around the neck and upper part of the chest for six days. Oxygen was administered in five minute periods every thirty minutes for five days and when needed for five days more. Oxygen gave so much relief that the child would cry for more to be given. The immediate use of oxygen in carefully graduated amounts probably saved the patient's life. The cough was distressing for a few days. Bloody expectorations lasted for about one week.

On the sixth day his condition was much improved; temperature, 99.6; pulse, 112; respiration, 40, with fair respiratory expansion of the thorax and fair respiratory murmur upon the left side but limited upon the right side.

A röntgenogram was taken upon the sixth day (Fig. 2).

No treatment was given the dislocated clavicle, because of his condition at first and later because reduction seemed impossible without an anæsthetic, and any general anæsthetic was considered contraindicated on account of the injured lungs.

The resulting deformity is slight and does not interfere with function.

A röntgenogram taken on the twenty-seventh day shows almost complete expansion of the injured lungs (Fig. 3).

His recovery seems practically complete at the end of four weeks.

It is very unusual for a victim of traumatic asphyxia to recover, because of the sudden complete interruption of the respiratory function which cannot continue any great length of time without death.

Traumatism, like the rolling and squeezing by crowds or compression by machinery, usually continues beyond the time of resuscitation.

If the patient could be quickly released from pressure and be given proper surgical aid at once, life might be preserved in many cases, but from the nature of the circumstances surrounding the injury efficient surgical aid could only be a rare coincident.

In this case the compression was of short duration, the patient was quickly carried into a hospital, where oxygen and efficient surgical aid were given at once.

#### SUMMARY

*Traumatic Asphyxia.*—This rare and striking discoloration of the face and neck, with conjunctival ecchymoses, accompanied by serious dyspnœa, following a compression injury of the thorax, are the four diagnostic symptoms of *traumatic asphyxia*.

*Rupture of Lung.*—Collapse of lung, extensive cellular emphysema, expectoration of frothy blood-stained sputa, following a serious compression injury of the thorax, indicate compression rupture of the lungs; unless physical or röntgenological examination shows rib fracture, when the lung injury may be due to penetration of the lung or pleura by the fractured rib.

*Dislocation of the Clavicle.*—This is definitely identified by physical and röntgenological examinations.

#### PYONEPHROSIS, URETERAL STONE

This woman, thirty-one years of age, a housewife by occupation, has been in poor health for a long time. She has a history of recurrent attacks of abdominal pain, lasting for three or four days, occurring over a period of several years.

Her appendix was removed one year ago, but without relief from the attacks of abdominal pain.

The initial symptoms of this attack occurred thirty-six hours before admission to the hospital. She suffered with severe, sharp, paroxysmal abdominal pains. The point of greatest severity of the pain was to the right of the umbilicus, and it radiated downward into the right thigh. Vomiting began immediately after the onset of pain and persisted until after she entered the hospital.

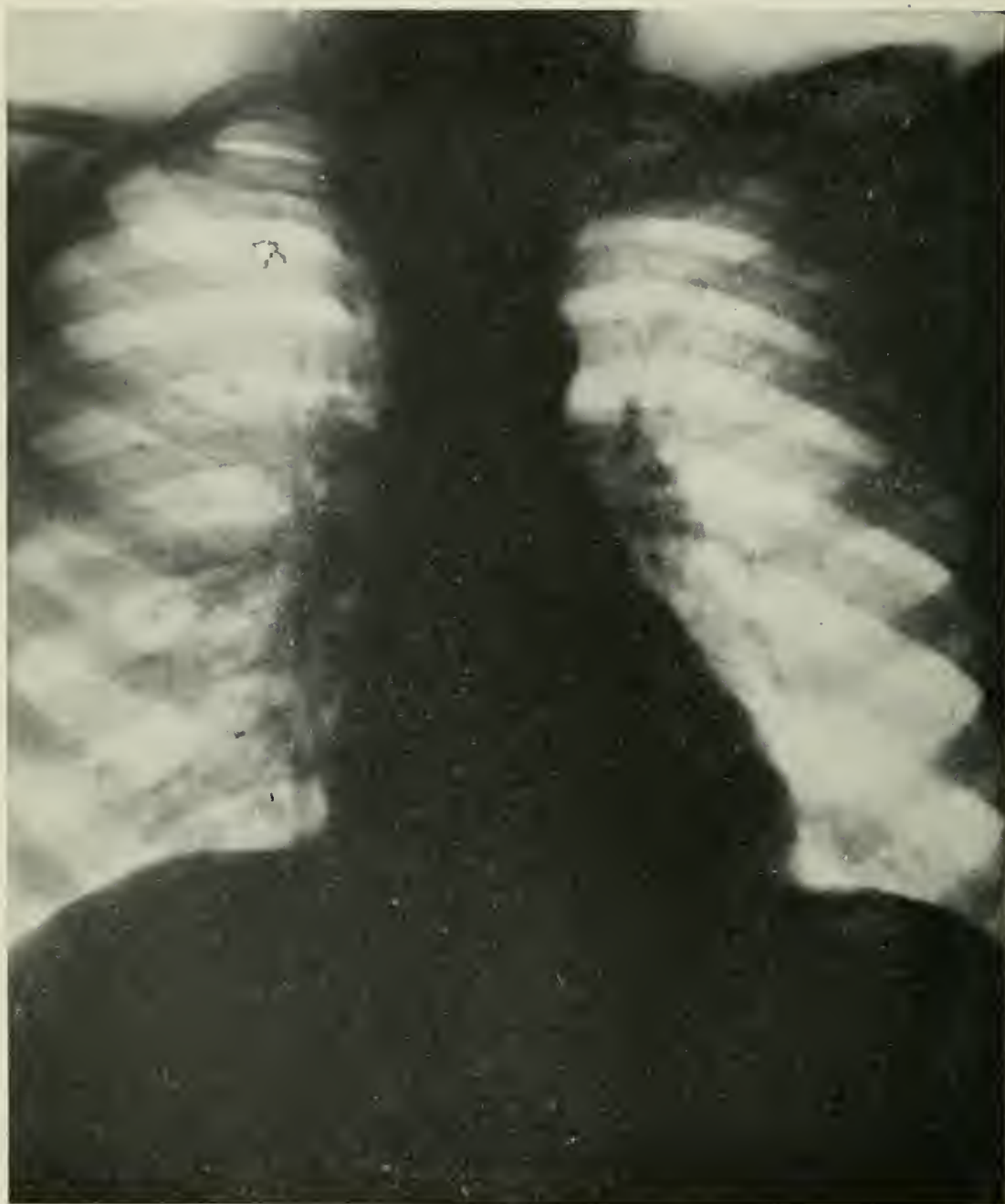
FIG. 2.



Case I. Röntgenogram of chest taken upon sixth day after injury. The right pulmonic field shows evidence of collapsed lung from the level of the second rib in the mid-clavicular line, seventh rib in axillary line and the sixth rib in the mid-scapular line upwards. There is marked increase in density just below the above-indicated levels, suggesting extravasated blood. The domes of the diaphragm are clear cut and smooth. The right appears to be pushed down further than usual as compared with left.

Summary of Röntgenograms. 1. Collapsed lung. 2. Extravasation of blood, right side.  
3. Emphysema, right side.

FIG. 3.



Case I. Röntgenogram of chest taken on the twentieth day after injury. No evidence of collapsed lung on right side. The dense shadow, suggesting extravasated blood, has disappeared. Pulmonary parenchyma can be noted in all portions of upper right side extending into apex. There is a line of thickened pleura extending from the right hilum downward and outward almost to the periphery.

Fig. 4.



Case II. Röntgenogram of the lower urinary tract, showing shadow of an oblong stone and smaller round one above it.

FIG. 5.



Case II. Röntgenogram showing opaque catheter in right ureter almost in contact with stone. The stone appears to be practically in the same axis as the catheter. The shape and position of the larger shadow are characteristic of a ureteral stone.

Examination at the time of admission to the hospital showed a woman acutely ill, practically in collapse, suffering from paroxysmal abdominal pain and vomiting.

Her urine was strongly acid ( $125^{\circ}$ ) and contained a few granular casts and some pus cells but no blood. Her leukocytosis was 22,500.

There was a tender palpable tumor in the abdomen to the right of the umbilicus. To differentiate between a tumor caused by an infected distended gall-bladder and a pyonephrosis of the right kidney, the colon was gently distended with air through a catheter in the rectum. If it were a distended gall-bladder the inflated colon would be below the tumor, but if it were a pyonephrosis the inflated colon would be directly in front of the tumor.

The inflated colon showed in front of the tumor, which was diagnostic of a kidney tumor. A röntgenogram showed an enlarged kidney without stones. A röntgenogram of the pelvis shows an oblong stone either in the bladder or in the ureter immediately behind it (Fig. 4).

Upon these findings the diagnosis of pyonephrosis of the right kidney was made.

The patient was operated upon a few hours after admission to the hospital. The right kidney was incised and drained. A small quantity of fetid pus was evacuated.

Without further operative procedure the patient was allowed to recuperate from the sepsis for about three weeks, until the urine discharging from the sinus in the right kidney was fairly free from pus. Apparently all of the urine from that kidney was being evacuated through the sinus.

The course of the infection, the quantity of urine discharged through the sinus, the shape and size of the stone shown in the röntgenogram (Fig. 4) together with its position, apparently posterior to and not in the bladder, indicated obstruction of the ureter by a ureteral calculus.

To demonstrate the location of the stone, a cystoscopic examination was made, the right ureter catheterized and a röntgenogram with the opaque catheter in position taken.

A No. 24 F. cystoscope was passed without obstruction. The bladder was found tolerant to instrumentation. The vesical walls

were everywhere slightly congested. The mucosa seemed somewhat thickened. No vesical stones were present.

The right ureteral orifice was surrounded by an area of inflammatory changes and marked œdema. The ureteral orifice was sunken in a pocket surrounded by the œdema. The orifice itself was somewhat patulous and pouting. There was a small flake of muco-pus attached to the wall at that point, which was easily displaced by irrigation.

This picture is practically pathognomonic of a ureteral stone lying near the orifice. The left ureteral orifice appeared practically normal.

Catheterization of the right ureter with a No. 5 F. opaque catheter met with definite obstruction, which could not be passed, at a point 3 to 4 cm. from the ureteral orifice.

A röntgenogram taken with the catheter in position in the ureter shows the top of the catheter in contact with the lower end of the stone (Fig. 5).

With the röntgenogram as a guide, the stone was located by bimanual examination in the broad ligament posterior to the bladder.

The stone was removed by operation through the vagina. An incision was made through the vault of the vagina, posterior to the cervix. The tissues were separated by finger dissection until the ureter containing the stone was located. It was carefully brought down into the vaginal incision and held in position, while the ureter was opened by a longitudinal incision over the stone. The calculus was then easily removed. The wound was left open for drainage, but there was no infection and no discharge of pus or urine.

To prevent stricture of the ureter at the site of operation, a longitudinal incision was made in removing the stone. There has undoubtedly been some injury to the walls of the ureter by the stone while it was imbedded in the ureter, with consequent cicatrization and contraction of the size of the ureter at that point.

The after-treatment in this case, to prevent stricture, consists in the catheterization and dilatation of the ureter at regular intervals.

In the röntgenogram (Fig. 4) there appears a small, round, opaque body not far from the ureteral calculus, suggesting another ureteral stone, but this is not the same shape or position of a urethral stone. It is round not oblong and not in the line of the ureter.

At the operation it was removed and found to be a phlebolith, probably from the pampiniform plexus.

#### HYPOSPADIAS

This little boy, three years of age, was operated for the penile form of hypospadias, eight days ago.

It is difficult to get good results with any form of operation for the relief of hypospadias in a young child.

The elements of difficulty are largely due to four things:

1. Urine soiling the field of operation during the period of healing.
2. Tension of the transplanted tissue, from strained position, deficient tissue, improper suturing or post-operative swelling.
3. Normal erection of the penis.
4. Post-operative interference with the repair by the child.

In this case the urine was side-tracked by a perineal, longitudinal urethrotomy, into which a catheter was secured. A curtain of oiled silk was placed between the catheter and the field of operation.

The boy had a large pendulous foreskin which was utilized as a transplant to form the new urethra. Sufficient skin was loosened from the foreskin to form the new part of the urethra. The flap was reversed and sutured with fine catgut so that a tube was made with the epithelium on the inside and the raw surface on the outside. The tube was made twice the size that was apparently necessary to give room for contraction. A large broad pedicle was allowed to remain attached to the prepuce to preserve the blood supply. The curvature of the penis was relieved by free dissection of the adhesions and bands producing it. A bed for the new urethra was made by lateral dissection of skin flaps from either side of the mid-line. The end of the urethra was dissected from its adhesions and its edges freshened so that it would unite with the tube of skin transplanted from the prepuce. The glans penis was perforated at the point where the meatus should be and the tube already prepared from the foreskin was pulled through and sutured to the end of the urethra. The lateral skin flaps were sutured to each other over the tube, with horse hair, without tension. The edges at the junction of the prepuce and glans were carefully denuded and sutured together to form the new frænum.

Since the operation we have tried to keep the wound dry and clean. The patient has been given bromides to prevent erections. His elbows have been fixed to prevent interference with the wound or dressings by himself.

The wound is yet clean and dry and the plastic work seems to be progressing favorably.

In another week the pedicle will be cut across and that part which is to form the edges of the new meatus will be trimmed to fit and sutured to the glans.

If failure occurs in any part of the plastic work, that part must be repeated until success has been attained, regardless of the time involved.

## ECTOPIC PREGNANCY

BY THOMAS H. KELLEY, M.D.

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GENTLEMEN: We have here Mrs. S., aged 32, a patient about to be discharged from the hospital. She has been married for sixteen years, never pregnant. Operated on fifteen years ago, resection of left ovarian cyst.

Menstrual history negative; last period July 6, 1921. First seen by us September 9, 1921, when pregnancy was diagnosed.

September 16th, again seen, complaining of so-called indigestion, pain high up in the region of the stomach. Had vomited twice; temperature, 99; pulse, 98. On the morning of September 17, 1921, she was brought to the hospital, and we found her pale, unconscious and practically pulseless, with all the signs of a severe internal hemorrhage.

She was operated upon soon after admission. The abdomen was found filled with blood and the right tube found ruptured with the head of a fetus, about 10 weeks old, protruding through the rent. Her condition was so urgent that all one had time for was to place two large 8-inch clamps at either side of the tubal mass, thus suppressing the hemorrhage to the pregnancy from both uterine and ovarian arteries. The patient was given cardiac stimulants and saline solution intravenously, before being returned to her room.

Reaction followed in due time, and in the course of a few days the condition of the circulation had improved so much that the tube was removed and the broad ligament sutured. This last procedure was rather unsatisfactory as the tissues were markedly indurated; the area was sufficiently drained. There was rapid improvement for five days, when weakness, faintness, sweats, gradual increase of the pulse rate, and escape of bright red blood from the incision, showed the hemorrhage was recurring. The patient was taken to the operating room again, and oozing discovered from the right cornu of the uterus and along the indurated surface of the broad ligament. A large Mikulicz drain was packed tightly against

the uterus, as well as in the cul-de-sac, and removed forty-eight hours later under nitrous oxide.

From this case, Gentlemen, you can see the feasibility of operating under the most unfavorable condition—a patient in shock from hemorrhage. With two hours of careful watching, her condition did not improve; operation was then decided upon.

We have a few brief records here of patients operated on in this Clinic in the last year:

CASE II.—Mrs. C. H., aged 34, one child living 12 years old; no pregnancies since. A rather large ovarian cyst resected some fourteen years ago. Patient first seen September 20, 1921, when she complained of vomiting, headache and missed period. On the 27th there was pain low down in the abdomen, rapid heart and nervousness. Fainted on the 30th; pulse, 120, and complaining of a peculiar “sticking” pain in the lower abdomen; no flow. Physical examination under an anæsthetic revealed a soft mass in the right adenexa. Operation (Oct. 21st) disclosed rupture of the right tube, with erosion on posterior surface at outer end.

CASE III.—Mrs. N. N., aged 26, married two years. Menses began at 16; of 3-day type, one or two napkins daily. First seen July 25, 1920, complaint of backache and severe cramp-like colicky pain in lower abdomen for last two or three days, but stopped as soon as menses began on this date. Defecation also painful. On bimanual examination no mass was palpable, but the tenderness was marked, out of proportion to the abdominal rigidity and distention.

July 30th, curettement, resection of ovarian cyst on right side. Unruptured ectopic pregnancy of same side also removed.

CASE IV.—Mrs. G. B., aged 34, five children, youngest 2 years of age. First seen, March 10, 1921, with pain in abdomen, cramps and continuous bleeding for past eight weeks, during which time she had been in various hospitals on four different occasions, and curettement done twice. Very anæmic; temperature, 99.8; pulse, 120. Bimanual examination revealed a rather large, soft mass in posterior cul-de-sac. At operation same day, uterus discovered to be bound down in cul-de-sac by dense adhesions. The left tube was degenerated and ruptured, being surrounded by a large organized blood clot. The tube on right side was also swollen and was removed, as it gave all the appearances of chronic infection.

CASE V.—Mrs. E., aged 35, married fifteen years, comes to us with the following history: Menstruation regular and negative until October 2, 1920, when two periods were missed. On December 3rd, began to flow again, and kept up continuously until the 17th when curettement was done. Remained in bed one week, the flow stopped. After being up and around for forty-eight hours, patient was suddenly seized with sharp, colicky pain in lower abdomen and fainted. Again remained in bed, this time for a month, during which time she had intermittent periods of flowing, with pain always in right side.

Examination January 18, 1921, in this hospital, revealed a painful mass in cul-de-sac and to the right. Diagnosis of ruptured ectopic made and operation the same day disclosed the pelvis filled with dark clotted blood, with a mass of adhesions in the cul-de-sac. This mass when raised proved to be the left tube and ovary, which were resected; tube on opposite side appeared macroscopically normal. Uterus small and infantile. A large denuded area was present on the bowel from adhesions to the cul-de-sac.

CASE VI.—Mrs. W., aged 35, widow with one child, 10 years old. First seen June 21, 1921, no signs of pregnancy—either subjective or objective; seemed worried about her condition. On the 25th, had slight cramp-like pain in lower abdomen, no flow; consulted another physician who diagnosed “appendicitis.” First period missed on the 28th.

July 8th, while in swimming, had sudden colicky pain in lower abdomen and was removed to her home. This severe pain continued until seen by us at 5 P.M. So long as the patient remained up and on her feet, the pain was not severe; but in the sitting or lying postures, very severe cramps arose. On account of this peculiar pain, bimanual examination was impossible. Board-like rigidity over lower abdomen; temperature, 99.8; pulse, 130. The diagnosis was “acute abdomen,” and an emergency laparotomy performed at once. On opening the abdomen, the peritoneal cavity was found filled with bright red blood and a fetus of about 2½ months free in peritoneal cavity. The cord led to the right tube which was removed.

CASE VII.—Mrs. T., aged 36, two living children, youngest 5 years, seen in consultation at hospital, August 22, 1921. Temperature, 99.6; pulse, 120; respirations, 18; leukocyte count, 15,000.

History of three distinct attacks of generalized abdominal pain in previous two weeks. These attacks which lasted from two to three hours, were always followed by vomiting, slight fever and marked prostration. Last one yesterday, when pain was most severe and vomiting had continued for succeeding twelve hours. Menses normal, 28-day type; last period August 10th, normal in amount and duration, no sign of blood since.

Physical examination showed marked tenderness over McBurney's point, with slight rigidity, and deep pressure over this area caused increased nausea. On bimanual examination the cervix was found of normal consistency, uterus apparently not enlarged. A firm mass, of rather large size was palpable high up in the right adenexa, and markedly tender on pressure (which also caused). The diagnosis was either appendicitis, ectopic pregnancy, or retention cyst of the ovary. However, at operation on same day, the appendix was easily found and apparently normal; gall-bladder negative. A definite hard, circumscribed mass palpable in right tube; ovary on same side slightly enlarged. The tube was removed, and on opening it a small fetus 2 centimetres in length, was found in the amniotic sac, unruptured.

CASE VIII.—Mrs. C. W. H., aged 32, married eight years; no children, no miscarriages. Period due September 10, 1921, missed; and the flow in the previous one was very scant. On the 15th, a few "sticking" pains in lower abdomen, accompanied by slight flow for two days. Pain returned on the 20th with nausea. First seen on the 22nd, when scant flow had begun with vomiting. Deep pressure over left iliac region reduced pain. Bimanual examination revealed the cervix softened, the uterus slightly enlarged, and a tumor mass—which was very tender—definitely palpable in left adenexa.

*Diagnosis.*—Ectopic pregnancy on left side. Patient refused operation, the flow and pain ceased after stay in bed two days.

Pain and flow returned on October 4th; she was operated on next day. A large, swollen, unruptured tube was removed, containing a fetus of ten to twelve weeks intact.

(This woman was operated in this Clinic two years previous, (Oct. 1, 1919), an emergency laparotomy being performed for

ruptured ectopic pregnancy on the right side, specimen showing a fetus of eight or ten weeks in the tube.)

From the above case histories, it may readily be seen how easily the condition can be overlooked. In one case seen at home late at night, the patient was not operated on until three days later. In two more, the condition was discovered accidentally during the course of an operation. In two cases referred, one was in a hospital on four different occasions, and had been curetted twice; another was allowed to remain in bed for a month following curettement before an operation was performed. On two occasions hemorrhage was severe; one of these patients nearly bled to death, requiring three different operations before recovery was complete.

With this in mind the dangers in delay before early operation due to faulty diagnosis can be readily understood. As there is no doubt, therefore, that a patient can succumb to hemorrhage, operation must not be delayed too long, waiting for reaction, even in such severe cases as the one of Mrs. S., this patient being operated upon during severe shock.

In the histories just read to you there was no one diagnostic feature peculiar to this condition. The symptom-complex, however, when complete renders diagnosis rather easy. If we are given a patient who furnishes a history of previous pelvic trouble, has passed a period or two, having cramp-like pains in lower abdomen, who has vomited and feels weak: one who begins to flow intermittently with a little fever and rapid pulse, is greatly relieved by rest in bed, only to have the foregoing manifestations return after being up a few days. When upon physical examination we find the woman pale and anæmic, with tenderness on deep pressure over lower abdomen—with some rigidity; while bimanual palpation reveals a soft cervix, slight enlargement of the uterus with a soft, boggy, painful mass on either side—the diagnosis is made without much difficulty. But unfortunately, these symptoms may not present themselves in so satisfactory a manner, and the diagnosis is, then, quite difficult. Only too often the uterus is curetted for a supposed abortion or the patient is treated for “stomach trouble,” “appendicitis,” and so on. There should be no difficulty in making a diagnosis of the acute variety of ectopic pregnancy. In the subacute one, however, the skill of the

examiner is sometimes taxed. It is in this type of case that the operation should be done at once to avoid the severe complications: Removing from the abdomen the tube affected, together with the fetus, placenta, the membranes and blood. In acute cases in extreme shock the operator will use his best judgement. In the first of the above reported cases with extreme shock, operation undoubtedly saved the patient's life.

The causative factor in ectopic pregnancy is, as a rule, not readily found. In going over fifty records from this Clinic, a definite etiologic factor could be demonstrated in about one-third of the cases. The commonly accepted causes are: (A) Congenital anomalies of the tubes; (B) external migration of the ovum; (C) obstruction of the tube from without, and (D) from within.

(A) It is not clear that this is a very potent factor, otherwise we should expect to find ectopic pregnancy in the first pregnancy as a rule.

(B) External migration of the ovum is disputed by many authors, at any rate it cannot occur very often.

(C) This is generally conceded to be the chief etiologic factor, arising from destruction of the epithelial lining through salpingitis which in turn is of gonorrhœal origin. This last affection is one reason given why ectopic pregnancies are more common in the centres of population, other things being equal, venereal diseases are more common in cities.

In the preceding histories, there was but one in which a previous suppurative condition of the tubes or ovaries could be demonstrated: Mrs. B., in whom the opposite tube was removed, the laboratory reports showing the presence of an old pyosalpinx, in the rest the macroscopic appearances of the opposite tube were normal.

(Probably no one cause is effective in all instances.)

In this series of 50 cases, 40 of the women were multiparæ, and in the remaining 10, some 4 gave histories of an ectopic following induced abortion. Two patients were operated on twice for an ectopic pregnancy on the opposite side; one three years later, the other practically two years later, as we have just heard from the case histories. This is practically the same proportion as in the large series

gathered by Smith. In a questionnaire sent to the personnel of the American Gynecological Society, he found of 1608 examples of tubal pregnancy, operated on by the members, there were 58 recurrences (3.6 per cent.) while in almost as many (1390) collected from the literature, there were 55 (4 per cent.).

As regards age, the average was 33 years, according with the usual incidence which is given as from 24 or 25, up to 33 to 35. Only one of our patients was in the 20's.

The number of years married, relation to previous sterility or normal deliveries, appears to have no particular bearing on the etiology.

In our series the pregnancy occurred in the right tube 26 times, and in the left, 14 times; in the outer half of the tube 21 times, and in the inner half, 19 times. While in the histories just read, there were 5 examples on the right and 2 on the left (the other double), there is no preponderance when a larger number are assembled. Thus in 742 cases alluded to by Schuman, the right was involved in 365, the left in 373 and both in 4.

Tubal abortion was the variety of rupture most commonly found by us. Of the other types, erosion proved to be more frequent than rupture from overdistension.

The average duration of the pregnancy was about seven weeks, with extremes of about three weeks and about four months respectively.

Pain is the most important evidence, as well as the most constant. It is present in practically all instances (80-84-95-and 96.6 per cent. respectively of four series referred to by Schuman.) Two varieties can be recognized as a rule. The sudden, colicky, cramp-like pain of the acute case, attended by excessive hemorrhage and shock.

The subacute type is by far the more common, however, and predominates in at least three-quarters of all cases. Here we have one or more minor attacks, the acute pain lasting from one to several hours.

Not all patients have pain of cramp-like character, but they will describe a peculiar "sticking" (pin-and-needle) sensation, situated deep in lower abdomen. We consider this a rather important point in the diagnosis of certain subacute cases. Patients are unable to

describe this peculiar variety of pain accurately; they will answer negatively to such questions as: "Is the pain colic-like as in intestinal cramps?" "Is it dull aching, boring, etc.?" It is the type of pain one might imagine would accompany a gradual slow distension of the tube, before an eroded area is so distended and ruptures that the internal pressure or tension is released. As the attacks progress, the pain is referred to one side of the abdomen, generally the same one as the lesion, and accompanied by chilly sensations, vomiting and constipation.

Hemorrhage from the uterus may be continuous or intermittent. It is usually associated closely with the attacks of pain, which it may precede or follow. In two of the above cases it was entirely absent; in another it was continuous for eight weeks. On going over the 50 case histories it was found the average duration of the bleeding was about 18 days, continuous in some, but intermittent in the great majority.

The cardinal symptom of the "skipped period" is absent in a great many instances, for example, only a little over one-half of Foskett's patients had missed. In two of the above cases it did not occur. Moreover, in the series of 50 cases, no less than 12 gave a normal, regular menstrual history. (This is of some importance in that too much reliance must not be placed upon it as an important feature of the symptom-complex.)

The temperature in this condition is, as a rule, not normal; on the other hand it is not much elevated. In the 50 cases, 2 had a temperature of normal or below. The average evening temperature (by mouth) was 100° F. Only one patient had a normal temperature throughout.

The pulse rate is usually of diagnostic import. In our series the range was from 80 to imperceptible; the average evening rate being 110. In 45 out of the 50 cases, the frequency was out of proportion to the temperature.

So, given a patient with abdominal pain, chilly sensations, vomiting, slight temperature (99 to 100° F.), with the pulse between 100 and 120, ectopic pregnancy should first be ruled out.

A leukocyte count is usually present. A blood-count was done in 38 of our cases and in 32 leukocytosis was present, averaging

13,500. The red count, of course, depends on the amount of hemorrhage; the lowest was 2,800,000. It is a general rule in these cases, that the lower the red, the higher the white-count, but the irregularity of these results is often confusing.

In differentiating an ectopic pregnancy from an inflammatory condition of the tubes or appendix, the most reliable sign in our experience has been the intense amount of tenderness, complained of on deep pressure over the affected tube. The sign is out of all proportion to the amount of rigidity, distension and fever present and can readily be determined by bimanual examination. The uterus in the great majority is in normal position, or can be readily placed there; since the pathologic processes have not been of sufficient duration for adhesions to form strong enough to fasten it in the posterior cul-de-sac. Palpation of a mass of soft and doughy consistency can be considered reliable only when the examiner is sure the cæcum and sigmoid are not distended with soft fecal matter.

After consideration of the foregoing facts we may look upon ectopic pregnancy as a subacute affection, one in which the symptoms continue—with one or more exacerbations—for one or more weeks; the patient in the meantime gradually becoming weaker and weaker until relieved by operation. The acute case of sudden onset, with fatal hemorrhage, is exceedingly rare; it is comparable, therefore, with the severe types of inflammatory condition of the tubes.

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# COLITIS—EXCISION OF COLON; BILATERAL DIRECT INGUINAL HERNIA—HERNIOTOMY; RECURRENT INGUINAL HERNIA—HERNIOTOMY WITH FASCIA TRANSPLANT

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## EXCISION OF THE COLON

CASE I.—Mrs. L. K., aged 38. Complains of distressing pain and tenderness in left hypochondriac region and down to left iliac fossa, a similar pain in the right iliac region; a severe colicky pain for a time before stools, which is somewhat relieved by a movement. Each stool is covered with or composed almost entirely of mucus. A few times there have been streaks of blood in the mucus and on three occasions a very marked hemorrhage of bright red blood. She has constipation—no natural movement, each time requires an enema—which returns in divided parts for one or two hours. Loss of appetite is so marked that she does not want any food; can take only liquids in small quantity. Has not for months been able to retain any solid food. Although she is on a limited diet, she has lost very little weight. There are frequent attacks of vomiting and dizzy spells in which she fears she will fall over and injure herself.

This case has had much trouble, including several operations in the last few years. Each time she has had temporary relief and each time the old syndrome, with perhaps a new symptom or two, recurred. She is married, has had two children, both are living and well. About seven years ago she had a perineorrhaphy and pelvic work done. Three years ago she had indefinite abdominal pains and digestive disturbance, which led to an appendectomy with very temporary relief. She was carefully studied and beyond a markedly ptosed stomach with some spasticity of the pylorus and with a slight amount of mucus in the stools, and a very slightly spastic colon, nothing pathologic was found, and for a time she was treated medically. To relieve the persisting stomach stasis a gastroenterostomy was performed which gave relief for a short time.

We find her at this time with the symptoms already enumerated and on examination a poorly nourished female with the skin pale, dry and sallow looking. Tongue is dry, tremulous and coated white. Pulse, 78, small, regular. Blood-pressure, 110-75. Urine: Light amber, clear; specific gravity, 1018; acid, no sugar, no albumin, no casts. Blood: Hemoglobin, 75 per cent.; red blood count, 4,200,000; whites, 7800.

Stools on culture show only customary flora; no parasites; much mucus, some pus and blood cells.

Barium meal and X-ray examination (made two weeks ago)—stomach, pylorus, antrum and duodenum normal in appearance. At six hours there is a slight residue. Twenty-four hour examination shows colon stasis, the meal being only half way down the descending colon. Forty-eight hour examination shows head of meal half way down descending colon and irregularity in filling of hepatic flexure and of descending colon. Cæcum fixed in all directions and with marked stasis. Hepatic flexure very tender.

Barium enema readily taken shows a large loop that fills just above the sigmoid, an irregular filling at and around the splenic and hepatic flexures and a mass collected at the cæcum. Abdomen is relaxed, flaccid, tympanitic; very tender all over the colon, but particularly at the cæcum, at the terminal part of the transverse, the splenic flexure and descending portions. Sigmoidoscopic examination shows a very much inflamed sigmoid and rectum with a great deal of mucus, but no evidence of ulceration.

It was felt that her colitis was sufficient to produce all her symptoms, either directly or indirectly, through a toxic or septic process. It was decided to operate with a view to removing the colon, but her general condition was not reassuring, because of her prolonged low feeding. On opening the abdomen we found a few adhesions from previous operations. The whole colon was filled with barium meal of seven days previous. The cæcum and ascending colon were very movable, the splenic flexure and upper part of the descending colon were fairly covered over from the lateral aspect with a membrane much resembling a Jackson's membrane. Efforts to remove this were rewarded with some success and the colon appeared to relax. Next, there was found an adhesion from the pelvis to the

sigmoid, producing a fairly sharp curvature at this point. This was severed and the sigmoid made free. Feeling that this would assist much to relieve the spasticity, obstruction and stasis, and in order to stop the passing of food over the irritated colon, an ileo-sigmoidostomy was performed.

The first step in this was to sever the ileum between forceps about two inches from the ileocæcal valve. The distal portion was ligated, inverted and buried by a purse-string suture. The proximal portion was similarly ligated and inverted. It was then brought around to the side of the sigmoid and a lateral anastomosis made between the two. I think the lateral is preferable to an end-to-side anastomosis, because it gives opportunity for a more even suture, a larger opening and less danger of stricture from contraction or from kinking caused by prolapse. As the condition of the patient was not good, closure of the abdomen was decided upon, hoping that this procedure would relieve the condition.

Again the patient improved and was able to take more food, but there was still a marked amount of pain in the abdomen. She has not satisfactorily convalesced during the week, and we have decided to complete the operation by removing the colon as the only means to give her relief.

*Operation.*—On opening the abdomen you note the light adhesions everywhere. The colon has much barium at the cæcum, splenic flexure and just above the sigmoid. This residue has been in the colon two weeks. The first step in removing the colon is to make free the cæcum and raise it up. A sufficient amount of peritoneum is stripped from the lateral surface of the ascending colon to cover the raw bed where the colon lay. The right colic vessels going to the cæcum and colon are now secured on the inner side of the bowel and the meso is severed, making free the ascending portion. The transverse colon is freed by raising the omentum, stripping its posterior layer from the anterior surface of the colon up to the upper border, ligating the branches of the mid-colic vessels that pass to the posterior surface of the colon and cutting the same. The two raw edges of cut omentum where the colon was removed are sutured, leaving a fairly normal omentum. The descending portion we treat as we did the ascending until we reach the sigmoid. Just above

the sigmoidostomy the colon is severed and the stump inverted. The colon is now wholly free and is removed. Now, we endeavor to peritonealize the bed of the ascending and the descending colon with the peritoneum saved from the lateral surface of these parts. It is to be noted that the ileosigmoidostomy is free; that there is a small opening at the gastroenterostomy and that there is no apparent tumor mass at any point. We fill the abdomen with hot normal saline solution to replace fluids lost and to decrease acidosis incident to the anæsthetic and the manipulation of operation.

I would draw your attention to the lack of evidence pointing to the colon at early operations; the focussing of attention on the stomach, gall-bladder and the appendix by the symptoms, and that not until the mucus appeared in macroscopic quantity was a diagnosis made and confirmed by the aid of the X-ray.

NOTE.—Following operation for several days there were attacks of vomiting at which times large amount of foul fecal smelling material were brought up. This necessitated washing the stomach on five occasions, and then a rapid convalescence followed, the patient leaving the hospital two weeks following operation. She was eating solid food, the first time in three years, and since going home has been on a general diet, retaining her food and gaining in weight and strength. There is still pain at times in the left hypochondriac region, but it is decreasing and will doubtless soon disappear.

This is a very interesting case because of the development over a long period of time, the lack of presentation of definite diagnostic symptoms, the temporary improvement after each operation done for the relief of prominent symptoms presenting at the time, which led physicians to conclude that it was a neurosis rather than an organic lesion; the partial improvement due to relief of the mechanical obstruction and the ileosigmoidostomy; and the complete recovery when the sluggish, thickened, inflamed colon was removed.

#### BILATERAL DIRECT INGUINAL HERNIA

CASE II.—J. B. comes complaining of a weakness in each groin with a marked swelling on each side, which passes down into the scrotum. There is no pain, but he is becoming more and more conscious as the months go by that it is drawing on his strength and decreasing his efficiency. He feels the need of supporting his

abdomen when he works, and at times, an urgency to decrease the size of the tumor.

Some twenty years ago he noticed a swelling, very small and replaceable, in the right groin, which has kept on growing to its present size. Only a few years ago he noted the swelling on the left side, but it is also growing. Both of these swellings are enlarged by standing or straining or by an increase in intra-abdominal tension. They are decreased, but not obliterated, by lying down or by taxis. They have an expansile impulse on coughing. They are soft and compressible in character.

There is no doubt that the diagnosis is inguinal hernia, whether direct or not, one can scarcely say, but the evident directness with which they come from the abdomen indicates that they do not travel any distance along the canal. They are neither of them femoral hernias because the neck of the sac lies internal to the pubic spine. In this case this is a sign that is easily discerned. The question arises what to do for this patient. It brings up the problem of the operable years for hernia. I am accustomed to, more or less arbitrarily, divide my uncomplicated hernia cases into three periods, and I want to emphasize that word uncomplicated, because the complicated cases come into a category quite separate from the uncomplicated. The first period embraces the years from birth to five, during which some appliance, particularly suited to the individual case should be used. I am very partial to a small compressible ball of yarn about 5 cm. in diameter, which is easily applied under a band and as easily kept in place. It is cheap and inexpensive enough that several may be had at one time. It is easily cleaned when soiled, and will usually serve the purpose better than more expensive pads. It is superior to a truss or appliance, which almost invariably irritates little ones, is not easily kept clean, is too expensive to replace frequently or to have several of, and altogether is not satisfactory.

If, at the end of five years, they do not thoroughly heal up, but recur, I think they should be operated.

The second period embraces the years from 5 to 65, which compose the active and productive years of life. During these years all cases should be operated that have no serious constitutional contraindications.

The third period embraces the years after 65. In this period only selected cases should be submitted to surgery and then usually under block anæsthesia. One cannot always be guided by the count of years, but rather by the constitutional age. Beyond this age the mortality or the morbidity following major operations usually offsets the advantages that may be gained from operation.

This man is 62 years of age with a temperature of  $97.6^{\circ}$ ; respiration, 18; pulse, 60, full, firm, compressible. Blood-pressure, 152-95. Urine: Specific gravity, 1018; albumin negative; no sugar; casts negative. He is a man who still has to earn his living, is vigorous and otherwise well, but feels very keenly the increasing weakness and draw on his strength. I think him a good surgical risk and so we will operate.

We will operate on the right side first. The usual incision is made parallel to Poupart's ligament two and a half inches long. This cuts through the skin, fat and fascia. The fibres of the external oblique are separated outward and upward from the external ring. The fibres of the internal oblique are seen arching over the neck of the sac. The neck of the sac is located and brought up, and the whole sac is separated from the cord. You will note how adherent the sac is throughout its length down into the scrotum. You will note that the cord lies external to the neck of the sac, and on top of the sac itself. You will also note that the neck of the sac is mesialward, very close to the spine of the pubes, and that lateral to it we find the deep epigastric vessels. We know now that we are dealing with a direct inguinal hernia and must conduct repair work accordingly.

As we proceed to remove the sac, we must bear in mind the possibility of the bladder being involved in the wall of the sac. This may be very easily opened inadvertently and, of course, if opened, must be immediately closed. It is wise now, with the peritoneal cavity opened, to inspect the femoral ring for a possible hernia. I had this impressed upon me during the last year when I was closing the inguinal canal by imbrication, after having excised the sac. The patient began to strain and a femoral hernia presented itself. It is unusual to find both inguinal and femoral hernias on the same side. I removed a few sutures, opened the neck of the sac and reduced the femoral hernia intraperitoneally, inserting a couple of sutures be-

tween Poupart's ligament and the pectineus muscle. Then I proceeded to close my inguinal canal. Finding no femoral hernia, we close our sac and proceed to repair our hernia.

In the direct type I do a modified Bassini operation. In the oblique I do an imbrication operation. Why? Recollect the anatomy and the part to be especially strengthened. In the direct type the sac has pushed through Hesselbach's triangle. It has produced a defect in the conjoined tendon. This conjoined tendon is to be repaired and supported. A transplantation of the cord makes this possible. You will note here that the conjoined tendon, which normally forms the posterior surface of the canal, is pushed aside. This we rebuild as well as possible by drawing together its edges. Next we open the rectus sheath and draw down the edge of the rectus muscle and with a few sutures attach it to Poupart's ligament. We now draw up the lower cut edge of the external oblique under the upper cut edge and suture it with a line of sutures at least one-half inch back from the edge. Then the upper cut edge is brought down on the outside of the external oblique and sutured. Now the cord lies outside all and just beneath the superficial tissue and skin. The skin is closed with silk sutures.

In the oblique type, on the other hand, it is much better to do an imbrication operation without transplantation of the cord for here we are desirous of reducing to normal the internal ring and of reënforsing this region. You will note that Hesselbach's triangle is very firmly supported and has no external ring in front of it as a weak spot, this having been obliterated by the duplication of the external oblique down to the pubic spine, or perhaps we should say transplanted to a point almost opposite to the internal ring.

*Left Side.*—Here we begin with a technic similar to that used on the right side. You will note here that the sac does not protrude to the depth of the scrotum, but that we must separate it from the cord and tie off just the same. Note the relations of the neck of the sac—mesialward the pubic bone, lateralward, the deep epigastric vessels, above the approximation of the external oblique and the conjoined tendon and below Poupart's ligament, all of which indicate that we have a direct hernia. Note also the absence of contents in the sac, and the fact that the bladder is not involved in the wall.

We will repair this as we did the opposite side—reforming

the conjoined tendon, drawing out of its sheath the rectus muscle for attachment to Poupart's ligament, transplanting the cord and imbricating the external oblique.

LATER NOTE.—Patient made a good recovery and left the hospital in eighteen days with a new sense of strength and of security in his inguinal region.

#### RECURRENT INGUINAL HERNIA WITH FASCIA TRANSPLANT

CASE III.—B.K. This is another case of inguinal hernia which presents a different problem. This man developed a hernia many years ago and was operated apparently successfully. About a year later he was thrown from his motor-cycle and noticed a swelling in the operative scar. He was reoperated and became infected. After draining for several weeks, the wound healed up and then in a few months he noted a recurrence of the swelling, the wound at no time feeling very strong. He was again operated, and following the operation had a large hematoma develop which, of course, prevented a primary union of the parts and as a result, he again has a recurrence.

On examination, we find a muscular, well-nourished, healthy looking young man, who has a large irregular scar in the right inguinal region. At the inner end of this, there is a mass which has an expansile impulse on coughing and is partly reducible. About an inch from the pubic spine there is an apparent deficiency in Poupart's ligament. One must approach this case with a certain degree of hesitancy. The number of operations that have been unsuccessful, the amount of scar tissue in the region are possible inherent reasons why union does not take place. A hemorrhagic diathesis and an imperfect Poupart's ligament are potential morbidity factors that must not be lost sight of, even if we consider the mortality risk of small moment.

It seems to me, that it is a case which ought to be tried again, and that a technic probably including fascia transplant will be the proper one. The transplant of fascia again adds another element of risk, but if successful, is extremely useful. It may be taken from the anterior and outer part of the thigh just below Poupart's ligament or if a much stronger transplant is desired, it must be secured from the tensor fascia femoris. This may produce some lameness in the thigh, which will eventually pass away. The transplant should not be removed until the hernia region is just ready to receive it.

We make our incision with a view to removing as thoroughly as possible all of the scar tissue of previous operations. As we cut the external oblique, we find the fibres are infiltrated with much new scar tissue. The internal oblique is quite thinned out, and beneath this we find the fibres of the rectus muscle attached to Poupart's ligament, showing a marked effort on the part of the last operator to produce a good result.

The sac is found protruding through Hesselbach's triangle. The cord is superficial to the sac and the deep epigastric vessels are external to the neck of the sac. Note that the deficiency in Poupart's ligament is quite marked but is not a complete one. The first point in repair is to obliterate this deficiency. This we do by removing the scar tissue and drawing together the severed edges. We now open the sac to be certain that there is nothing in the same, and also to assure ourselves that the ligature around the neck of the sac does not include the bladder. It is so easy to open the bladder in direct hernia that a reiteration of a warning at this point is worth while. The sac is now tied off and excised. Now is the time and this the place to insert the fascia transplant. An incision three inches long in the outer anterior surface of the thigh is made down to the fascia lata. The skin and fat are reflected to uncover an area about two inches by three inches of the fascia. This incision and reflection must vary in size according to the needs of the case. Sometimes a much larger transplant than the above is needed. Always we must remember that tissues contract about one-third, and exercise care that the immediate contraction does not leave the transplant too small to cover the weak spot.

The transplant is now placed next to the peritoneum and sutured to the under surface of the conjoined tendon, the rectus fascia and pubes, Poupart's ligament and laterally to the region of the deep epigastric vessels. The conjoined tendon is now brought down and sutured to Poupart's ligament. The rectus sheath is opened and as an extra precaution the rectus muscle drawn down and out and sutured to Poupart's ligament. The lower cut edge of the external oblique is now drawn up under the upper edge and sutured about one-half inch from the edge with a few mattress sutures. The cord is now placed on this layer and the upper edge drawn down and

sutured to the outer surface of Poupart's ligament and the external oblique. This must not be sutured too tightly; this gives us a support of the weak spot in Hesselbach's triangle, of peritoneum, transplant, conjoined tendon, rectus muscle and two layers of external oblique.

Some of these transplants will not take just as we find some skin and fat or Thiersch grafts do not take. Unfortunately, when this occurs results are seldom satisfactory and in some cases the whole graft will slough away. There appears to be less tendency for this to occur where the graft is placed next to the peritoneum. The greater body warmth, the more liberal bathing in serum, the better nourishment in this location as compared with the intramuscular and interfascial planes have probably much to do with this greater success. You will note the great tendency to capillary bleeding in this case. It is very difficult to check, and I am wondering what effect it will have on the healing of the wound. In order to minimize it, we will put on a dressing which I use frequently in hernia cases. An inverted pyramidal dressing is applied over the wound. A strip of adhesive about thirty inches long and three inches wide is now applied. Beginning just below the 12th rib attach the adhesive in the direction of the wound; flex the limb at the hip and draw the adhesive tightly down over the wound and groin to the inner side of the thigh and then around in the fold of the buttocks. When the leg is extended additional tension is added. This is uncomfortable for twenty-four hours, but it protects during the period of straining. At the end of twenty-four hours or even twelve hours, it is cut in the fold of the buttocks and the tension relieved.

I consider it superior to the spica because it is so simple, so much more easily applied, and produces tension and pressure exactly where desired.

LATER NOTE.—A large hematoma formed in the thigh wound which caused much discomfort. The inguinal region became markedly infiltrated, but no definite localized hematoma could be outlined. Blood examination showed coagulation time eight minutes. Recovery was uneventful and the immediate results were good. Patient stated that the region has a much greater sense of security than ever before.

## PELVIC INFECTIONS

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IN writing this paper we do not expect to offer anything new on the subject of pelvic infections; merely to classify them in order that we may more intelligently treat them.

Gynecologists are quite agreed upon this subject but their views are very often at variance with those of the general surgeon. The classification is general, as each case is a law unto itself, and certain obscure cases tax the judgment of the most experienced operators.

It is very essential, in the first place, to know the pathology of the different structures involved, as well as something about the invading organisms with which we have to deal.

The organisms of importance most frequently found in pelvic infections are the gonococcus, streptococcus and the tubercle bacillus. Often we have a combination of these organisms and not infrequently some of the ordinary pus-forming organisms in addition. Mixed infections are quite common and are more resistant to treatment.

We shall begin by speaking of the least common infection, that of tuberculosis. *Tuberculosis of the tubes* attacks both the mucous membrane lining the tube (endosalpinx) and the peritoneal covering (perisalpinx). This structure is in an especially favorable location for the growth of the bacillus of tuberculosis on account of its convoluted form and of its succulent lining membrane. This type of infection, according to Graves, is usually bilateral. It is either ascending or descending and it is very doubtful whether it is ever primarily imposed upon the tube. This form of infection may follow the blood stream from some distant focus, in which case it is known as a hæmatogenous infection.

Infection from tuberculosis of the external genitals rarely, if ever, ascends, and in this respect is in marked contrast to the course of gonorrhœal infection. On the other hand, descending infections originate from tuberculosis of the peritoneum or from the intestines about the tubes, in which case either perisalpinx or endosalpinx may

occur. When endosalpinx develops the disease becomes specialized. The hæmatogenous mode of infection in the tubes is usually a metastatic growth of tubercle bacilli which has come through the blood stream, most frequently from some distant focus, such as the lungs. In this case the original area of infection may become healed and the tubal infection appear to be primary, although in reality it is secondary. Unless it happens to be a part of an acute miliary tuberculosis, the disease is chronic. The condition resembles that which is of gonorrhœal origin. The content of a tuberculous pus-tube consists of a white mushy cheese-like material, unless, as is quite common, a mixed infection is present, when it assumes the character of a gonorrhœal tube. In fact, a gonorrhœal infection may also be present.

A tuberculous tube is often discovered only by a microscopic examination. An old tuberculous infection is either caseous or fibrous, although sometimes a hydrosalpinx forms. A perisalpingitis of tuberculous origin can usually be recognized by the presence of the tubercles on the peritoneal surface of the tubes as well as by the appearance of the adjoining structures. Tuberculous tubes are nearly always associated with dense, strong adhesions; much more frequently than with gonorrhœal infections.

The tubal symptoms unassociated with general peritoneal involvement are about the same as in gonorrhœal salpingitis. The findings on bimanual examination are about the same in both cases, and unless tuberculosis is quite evident elsewhere, the diagnosis probably will not be made. The progress of tuberculous infection is slow and sterility is almost inevitable. The treatment does not vary greatly from that for gonorrhœal infections, except that more often a radical operation, such as hysterectomy should be performed. Conservative operation is most apt to be followed by a recurrence of the disease. Some of these cases are inoperable, such as those where a densely adherent mass is present, which involves the bowels. The patient, however, through proper hygienic measures may make a good recovery. Tuberculosis occurs in the uterus in about half the cases where the tubes are involved. The ovaries not infrequently become infected with the tubes by the hæmatogenous route, but not often are they primarily infected.

The streptococcic infections of the uterus and tubes usually follow abortions, miscarriages, puerperal sepsis or instrumentation of any sort. There are a few cases, however, which are not due to this puerperal sepsis. The onset of the infection at such times is severe and frequently causes death. In the cases that survive, few adhesions are formed and it might be likened to an attack by the enemy where the defense is unprepared, there being no time either to build a wall or to dig trenches. The victim either succumbs at the time of attack or if she lives, the structures within the abdomen exhibit only slight evidence of the invasion, sometimes the organisms lie dormant for months and years and it is best to leave them that way. These cases we should not operate upon, since it may happen that the organisms become active and a new infection occurs and the patient dies. Polak mentions four cases, and I have had one, which gave a history of having had a streptococcic infection a few years preceding her present illness which developed a streptococcic peritonitis when operated upon and subsequently died. Streptococcic infections usually spread by means of the lymph channels, causing infection of the broad ligaments and of the side walls of the pelvis and about the cervix. We regard the clinical record in pelvic infections as vital in importance and our main reliance in making a diagnosis.

We now come to gonorrhœal infections, the most frequent and the most common. We shall speak very briefly of the external manifestations. The majority of the infections occur first in the urethra. Graves says from 60 to 90 per cent.; perhaps the percentage would be higher except that the symptoms are hardly severe enough in this structure to attract the attention of the patient.

Skene's glands, the small secretory structures situated on either side of the urethra, may be primarily attacked, but usually are infected after the urethritis. The infection of Bartholin's glands is next in frequency but the vagina, not having glands, escapes any serious involvement.

We can now consider the cervix. From this point on we are mostly concerned with the pathology, since our treatment of pelvic gonorrhœal infections is based upon the pathology from the cervix upward.

In this connection it is necessary first to emphasize the structure

of the cervix and of the body of the uterus. It is well to consider the fundus and the cervix of the uterus as two distinct organs which differ from each other more or less essentially in their histologic formation, physiologic functions, and in the manner in which they are affected by germ invasions.

The mucous membrane of the cervix is quite different from that of the body of the uterus. In the endocervix the membrane is composed of complicated Racemose glands with small ducts emptying into the cervical canal. The epithelium lining the cervical glands is of the high cylindric, goblet-cell, type. These glands secrete true mucus. The endocervical mucosa is thrown into definite rugæ radiating from a central line in an arrangement known as the *Arbor-vitæ*; these rugæ or folds are of clinical significance in inflammatory processes of the endocervix.

Toward the internal os the mucous membrane becomes modified, the glands become less arborescent and the high cylindric cells become lower in type. At the internal os the mucous membrane of the cervix merges into that of the endometrium.

The mucous membrane of the endometrium is composed of simple tubular glands lined with a low cuboidal epithelium and secretes a thin watery fluid which is not true mucus. Thus, while the endometrium and endocervix differ histologically and in their physiologic function, they also differ in their response to pathogenic organisms.

Normally the internal os acts as a barrier to all organisms except the gonococcus, tubercle bacillus and the spermatozoon. If, however, the os is partially dilated or if it becomes patent as a result of parturition, infection is possible from many pathogenic organisms. Thus in the non-impregnated uterus, ascending infection in the endometrium and in the tubes rarely occurs except from gonorrhœa. The endocervix, however, is more susceptible to infection as the external os affords less protection and the mucous folds of the cervical mucosa constitute a favorable soil for various organisms.

The gonococcus, especially, often remains latent, either in the folds of the lining membrane or in the ducts of the glands, ready to become active under the hyperemic influence of menstruation, parturition or coitus.

It is highly essential that the endocervicitis be treated and an

attempt be made to cure it before a laparotomy is performed. We may curette and cauterize the endocervix, open retention cysts, but if the infection is severe we should amputate the cervix, but we should never curette the uterus as we have already shown that it is the dilatation of the internal os that spreads the infection upwards.

Curettements of the uterus are useless and even harmful.

The infected endometrium of the corpus uteri, when involved only slightly, is not a serious matter. The true acute gonorrhœal endometritis is rarely seen and we must conclude that the endometrium serves principally as a path to the tubes for the gonococcus and that it does not furnish a good soil for the growth of this organism. When infection does occur, recovery is rapid and due to a good blood supply and to a good drainage.

An immediate infection of the tubes following urethritis and endocervicitis is uncommon since as we have said the internal os forms an efficient barrier, hence it takes months and sometimes years for latent gonorrhœa of the endocervix to cause pus tubes.

The tubal isthmus, though small in calibre, permits ready infection of the tubal mucosa, practically both tubes are always infected as gonorrhœal salpingitis is originally bilateral and both tubes may heal or one tube may heal while the other may form a pus tube. Unless this bilateral invasion is kept constantly in mind at the time of operation, we are apt to leave infected tissue which later on will undergo an acute exacerbation. We have all seen cases in which one tube was grossly involved, while the other apparently was free from disease, nevertheless, during convalescence or during the patient's stay in the hospital, the other tube became a swollen and tender mass.

It was formerly supposed that no accumulation of fluid could occur in the tube until both ends were sealed; but in serial sections through the isthmian and through the interstitial portions of the tubes Polak and others have demonstrated in a large number of cases that the uterine end never closes and that the apparent occlusion in the isthmian and the interstitial portions is relative and not actual. This may be explained by the fact that the folding, the œdema and the swelling of the mucosa make the actual lumen so tortuous that the intratubal pressure closes or nearly closes the uterine end.

The interstitial portion of the tube is surrounded by an inner

circular, muscular layer which is continuous with the circular muscular coat of the uterus and as a consequence infection here must naturally excite inflammatory reaction in the submucous and muscular structures immediately surrounding the mucous tube.

The muscular coat, when examined microscopically, shows œdema and infiltration with inflammatory tissue cells and consequently in mixed infections, when the streptococcus is present, small abscesses are found in the muscular tissue.

The small round-celled infiltration causes an increase in the formation of the connective tissue of the uterus. This in turn causes an enlargement which does not subside with the removal of either one or both of the tubes. The persistence of this infection of the uterine muscle is manifested clinically by persistent leucorrhœa and by the formation of adhesions of the tubes, bowels and of the omentum to the fundus. How often we have opened the abdomen after previously incomplete operations for tubal infections and have found many adhesions along the fundal portions of the uterus. Clinical experience reveals this condition a great many times. In fact, such a large percentage of cases which came for operation to the County Hospital had previously had one, two and sometimes three, operations upon the pelvic organs that we took it upon ourselves to look over a thousand cases of women who had been operated upon for pelvic infections.

The facts ascertained were extremely interesting. Of the one thousand cases operated upon for salpingitis 138, or 13.8 per cent., had been operated upon at some former time for a pelvic condition which as nearly as we could determine was about the same condition for which they now presented themselves.

These figures are almost certain to be too low since without doubt many cases preferred to continue as they were rather than to submit to another operation. The death rate in this series of 1000 cases was 2.5 per cent. and the average age it is interesting to note was  $25\frac{1}{2}$  years.

It is of interest to note the effect of gonorrhœal infection of women upon the human race. Norris says "it is the most potent factor in the production of involuntary race suicide and by sterilization and abortion does more to depopulate the country than does

any other cause." Polak found in a study of 789 cases of sterility that over 400 proved to be so as the result of chronic pelvic inflammation, in which a history of gonorrhœal infection was definitely obtainable and local lesions demonstrated.

In France, where statistics have been accurately compiled it has been found that of about 10 million families, 2 millions are without issue; these results according to Neisser would tend to show that gonorrhœa is the etiological factor in nearly one million sterile marriages and this does not include the vast number of "one child sterilities" due to this condition. It has been shown that the birth rate in the Negro race is diminishing. If I were to venture an opinion not based upon actual figures but upon experience in the operating room upon a large number of colored women, I would say it was principally due to gonorrhœa.

Having reviewed the pathology of gonorrhœa infections we may now proceed to the question of treatment which we feel is both rational and conservative. The treatment is classed as conservative and radical. The conservative treatment is not operative. A certain small percentage of these cases recover, or, at least, even though masses remain in the pelvic region, they are free from symptoms. Among the well-to-do patients more acute exacerbations may be allowed to occur before resorting to operation inasmuch as the loss of time and the expensive nursing, etc., can be borne better by people of this class. Among the indigent another situation arises. If patients have had two or more exacerbations of pelvic inflammation we decide upon operation. Should pelvic abscesses occur we drain it per vagina.

The time of operation is chosen only after the temperature of the patient has remained normal for several days and when the blood count is below 11 m. The reason for this delay is that the tube contents should be as near sterile as possible. We base our operative procedure upon existing pathology and also upon the age of the patient.

The operation of hysterectomy, or the more conservative procedure, depends upon the condition of the ovaries. We feel that the operation of choice in younger women with one or both ovaries in good condition is the Bell-Buetner operation, or what is sometimes called the high amputation operation. This procedure has the advantage of removing the pathology and preserving a circulation of the

ovaries as well as that of maintaining the menstrual function for the patient. We feel that it is highly desirable not to interfere with the menstrual function in young women.

Where both ovaries without any question are diseased, we do a hysterectomy coring out the cervical stump in order to remove the endocervix. This operation is done irrespective of age when we feel that it is better to make a clean sweep than to leave diseased tissue which we know will cause trouble eventually and necessitate another serious operation.

When the cervix is lacerated as a result of childbirth or where it has resisted treatment, or where there is the slightest suspicion that there is a beginning malignancy, we do a complete hysterectomy and save one or both ovaries or none at all according to their condition.

At times, we feel obliged to save an ovary that is more or less diseased when the patient is young, even though this ovary may later undergo a cystic degeneration, becoming large and painful and require subsequent removal.

These cases try the judgment of the most experienced operator and when in doubt and the woman is young, we save the ovary. With the older patients, when uncertain, we prefer to remove the suspicious ovary.

When the patient is near the menopause, we have fewer symptoms if all the diseased tissue is removed, even though the ovaries are resected, than if we leave diseased tissue.

Graves and others have said that we have fewer post-operative symptoms after hysterectomy now than we had formerly. This is probably because we leave less diseased tissue for the subsequent formation of adhesions and also because they cover the raw surfaces better with peritoneum.

Practically we never drain these cases as we did formerly, since we feel that the pus is sterile to begin with. At first it took considerable courage to close a case which had free pus in it, but as yet we have not had cause to regret it. If the case is more acute than desirable for operation and much pus is present, possibly we would drain.

The hysterectomy which we use is one which we have simplified and is performed as follows: After the removal of the uterus in the ordinary manner we use the following technic. We first place a

double number two chronic catgut suture through the cervical or vaginal stump and bring it out and through the broad ligament so as to include the uterine artery as illustrated in Fig. 1. We then place a similar suture on the opposite side. These two sutures take care of the uterine arteries as well as the small bleeders in the stump. The next step is illustrated in Fig. 2 and is as follows: Using a double number two chronic suture about eighteen inches long we place the suture through the broad ligament to include the ovarian artery. This suture is continued through the raw edge of the broad ligament to the centre of the cervical or vaginal stump and then brought back in such a manner as to include the round ligament. The same suture is then again placed through the stump so that when the suture is pulled tight, the round ligament is brought down to the centre of the stump. The long end of the suture is then tied to the end of the suture which encircles the ovarian artery. This procedure puckers the broad and round ligaments into the cervical stump as illustrated to the left in Fig. 2. The next procedure then is to cover the raw surface with peritoneum. This is done by means of the Rossini procedure which is a part of the procedure used in the Bell-Beutner operation, or what is sometimes known as the high-amputation operation. By means of this procedure we suture the bladder over the raw surface as is illustrated in Fig. 3. We include only the peritoneal coat of the bladder, which we suture to the posterior wall of the vagina. This operation for hysterectomy not only gives good support to the pelvic floor but it holds up the vagina as it should be and incidently cures a moderate degree of cystocele. This procedure in itself could be described separately as a distinct operation for the relief of a moderate degree of cystocele. The operation is very much easier and quicker to do than the ordinary operation for hysterectomy. We also wish to speak about the diagnosis of pelvic condition by means of the X-ray.

Visualization of the pelvic organs has now become possible by means of the X-ray. We are all familiar with what the X-ray means in diagnosis to the general surgeon and to the internist. It has formerly meant very little in the diagnosis of gynecologic conditions.

It is possible by the use of gas injected into the peritoneal cavity to obtain X-ray pictures that show the relationship to one another

FIG. 1.—First step.

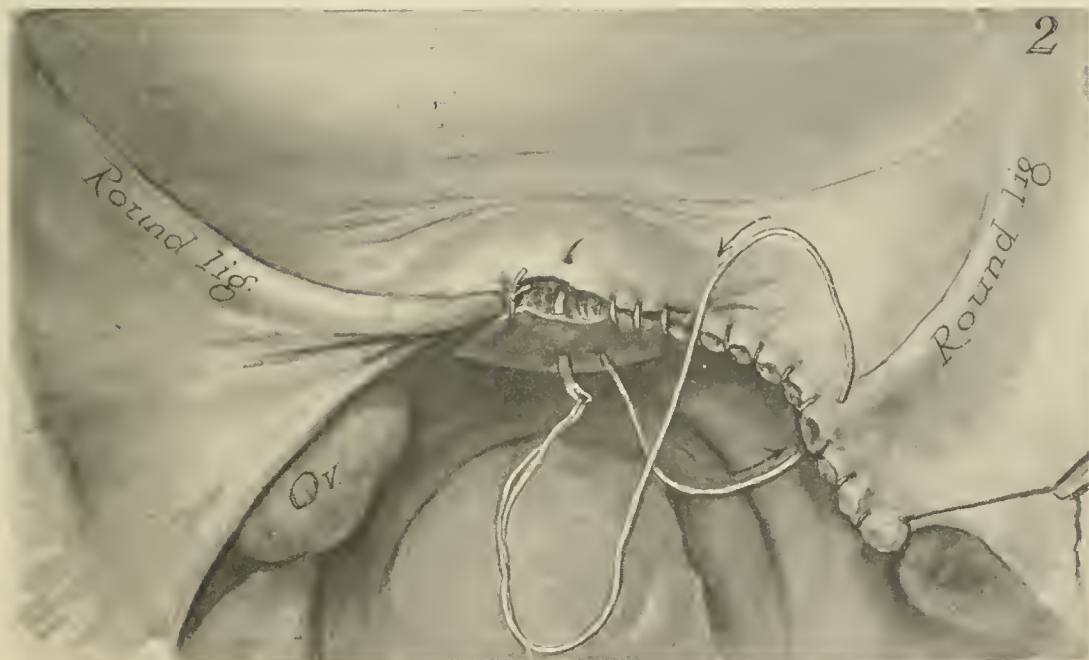
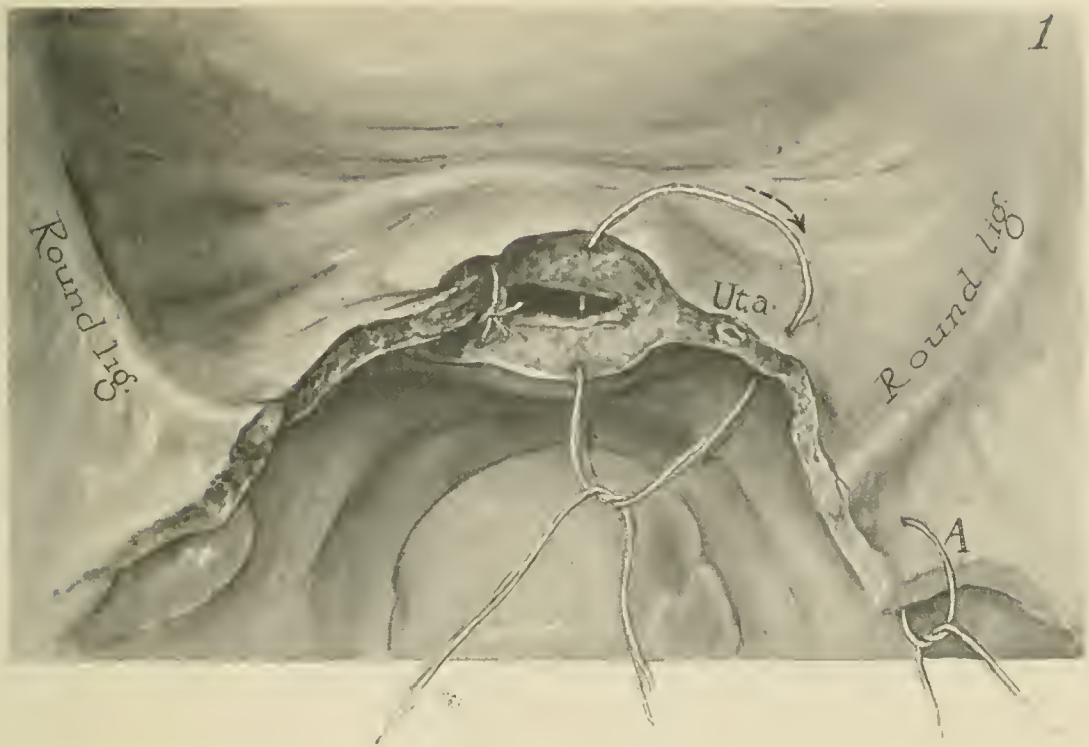
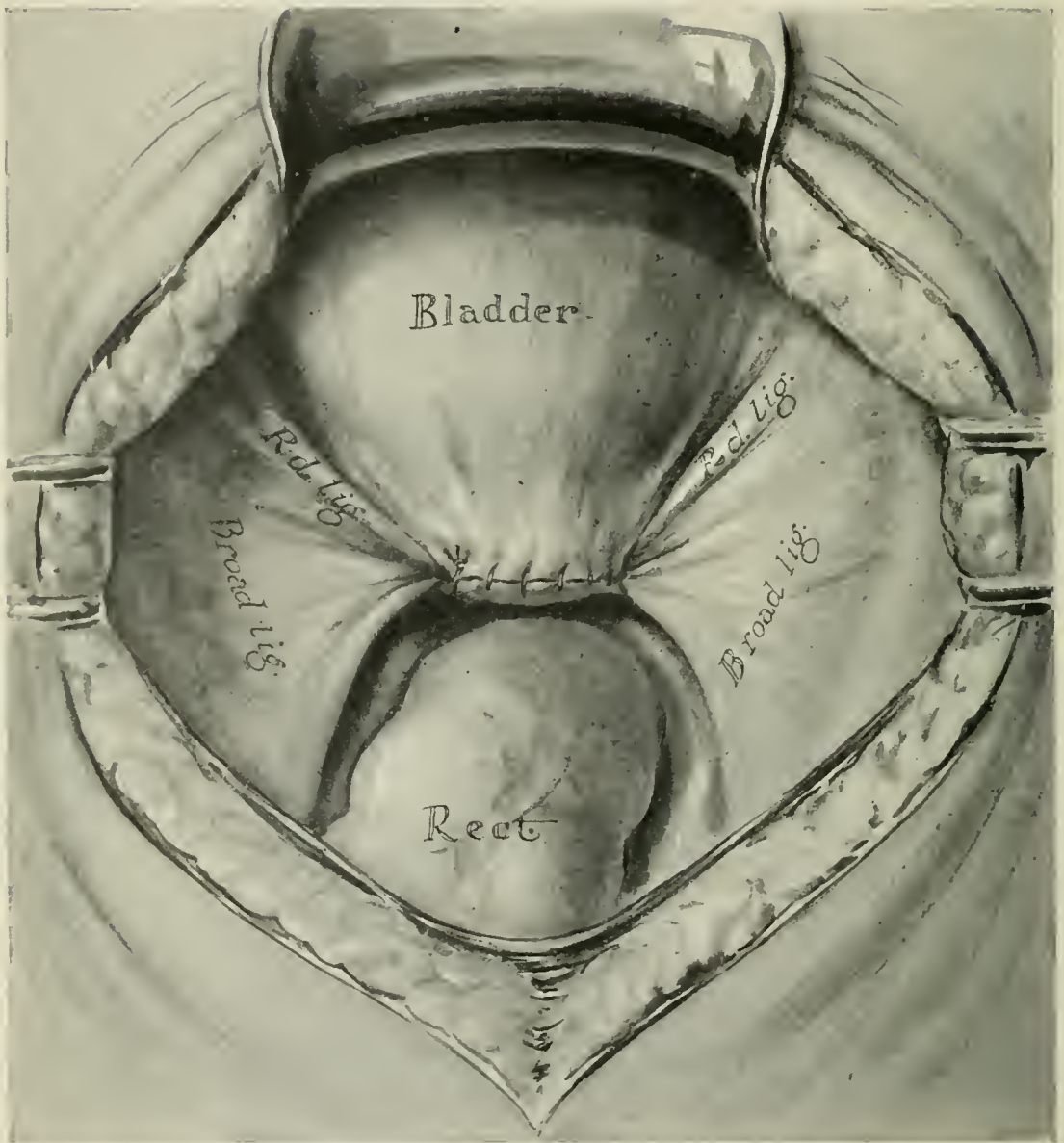


FIG. 2.—Second step.

FIG. 3.



Suturing the bladder over the raw surface.

of the pelvic organs, as well as the presence of adhesions and foreign growths. These pictures aid in checking the physical findings, especially where we have to deal with a very thick or rigid abdominal wall. We all have cases where we are unable to make a satisfactory bimanual examination due to various causes.

Anything that will aid or tend to make more exact the diagnosis of these conditions is worthy of consideration.

This method is by no means a routine procedure as many cases are very easily diagnosed by the ordinary examination. Any procedure must be safe and easily applied, otherwise it would be of no value. If the cases where it is to be used are selected with care, it is safe and very easily applied.

We use carbon dioxide gas in preference to oxygen because the former is more quickly absorbed and as a consequence is less painful. Carbon dioxide gas is usually absorbed in about a half hour, whereas oxygen may remain in the abdominal cavity for forty-eight hours before it is absorbed. This fact has been proven in operations as we have found oxygen gas present two days after it has been injected. The gas is sterile apparently as it comes to us in the tank. In testing the gas the laboratory findings have been negative for infectious organisms.

If the patient has an acute infection we feel that it is a contraindication for its use.

We inject the gas by inserting a needle through the abdominal wall, into the peritoneal cavity, or we may pass a Rubin cannula into the cervical canal. If we have infection of the pelvis, we use the former method. If we have no infection, we use the uterine route. This latter method is of diagnostic importance in testing the patency of the tubes. Instead of assuming in the absence of apparent pelvic inflammation that sterility is due to a constricted cervix, we pass the cannula into the uterine canal and determine by gas pressure the patency of the tubes. If the tubes are open the gas passes into the peritoneal cavity. Of course, it is hardly necessary to mention the fact that if pregnancy is suspected, we use the abdominal route. If adhesions of the bowels and of the anterior abdominal wall are suspected, it is well to go to one side of the median line as in making an incision following a previous laparotomy, when we make our in-

cision to one side or the other side of the old scar. We otherwise insert the needle about an inch and one-half below the navel in the median line. As a preliminary we flush the patient's bowels and have the bladder empty. We give the patient a dose of scopolamine and morphine about one-half hour before injection of the gas. One-fourth grain of morphine and 1-150 grain of scopolamine is sufficient. We do not use a local anæsthetic in the skin, because we feel that the needle used for such a procedure is as painful as is the needle which we use for the gas inflation. We use the ordinary needle that is used for spinal puncture. After the needle is inserted, we allow the gas to flow into the abdominal cavity. The gas is passed through a bottle containing sterile water before inflating. We open the valve of the gas tank and by means of a Tycos manometer, we gauge the pressure which usually runs about forty to sixty mm. of mercury. If the needle is not in the abdominal cavity, or is obstructed, the indicator immediately registers much higher and means that we must readjust it.

As the gas enters the abdominal cavity, the patients usually complain of pain in the epigastrium which radiates to each shoulder. This pain lasts but a few minutes. Gas measuring devices are on the market but one can easily judge how much gas is required. Each patient is a law unto herself as a large stout person requires more gas than a small one. The abdomen becomes moderately tympanic. After the needle has been withdrawn from the abdomen the patient is sent immediately to the röntgenologist. The length of time of exposure depends upon the size of the patient; usually ten seconds is sufficient.

The patient lies upon her abdomen with the hips raised, and a picture is taken through the outlet of the pelvis. The patient is then sent back to her room and, as a rule, she experiences no further pain. As a matter of fact, they usually sleep as a result of the dose of scopolamine and morphine.

We have used the procedure in about sixty cases thus far and have had no untoward results. We have, however, received some very useful information relative to the pelvic condition.

# Medicine

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## HYDATID CYSTS OF THE LUNG

WITH A REPORT OF FIVE CASES

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ALL clinicians have noted that certain rare affections will occur in series within a short time. Thus, I have had the good fortune to successfully meet with five cases of hydatid cysts of the lungs within the lapse of four months, and given the relative rarity of this morbid process, I believe that a detailed report will not be devoid of interest.

CASE I.—*Suppurating hydatid cyst at the base of the right lung. Hemoptysis. Diagnosis by puncture and radioscopy. Small repeated vomits. Pneumotomy. Recovery.*

Female, *æt.* 9 years, entered hospital October 6, 1916. The child was born at term and has never been ill. Began to walk at the age of fourteen months. Father and mother in good health. Seven brothers and sisters of the patient, all in good health. No dog lives in the house occupied by the family.

A year ago the patient accidentally fell into a pond and the parents state that since this occurrence the child's health, which had been previously excellent, commenced to decline. She has become pale, with loss of appetite and has emaciated. A physician was consulted and after auscultation an exploratory puncture was made on the right side of the thorax behind, and as some clear fluid was withdrawn the physician suspected a right-sided pleurisy and ordered the patient to enter hospital immediately.

The child was admitted to hospital December, 1914, and remained four months. Since the first puncture was made the patient coughs and expectorates, but there never was hemoptysis. At this time there were signs of a fluid collection at the right pulmonary base. Weinberg's reaction, done on January 15, 1915, was negative. Exploratory puncture withdrew a perfectly limpid fluid and the parents

were informed that a small cyst of the lung could be cured by operation, but they preferred to take the child home. At the time she left the hospital the general health had improved, the appetite had returned, but there was a disgust for meat and fats.

Six months elapsed, when in the evening the child had a slight hemoptysis and from this time on hemoptyses succeeded each other at irregular intervals and with varying severity. Symptomatic treatment was essayed by several physicians who were consulted but without success. The child's health did not decline much, but during the month of October, 1916, there was such a severe hemoptysis that the patient was at once admitted to hospital on October 6. On the following days the temperature went up to 100.8°F. in the morning and 102.7° in the evening and this continued until October 14.

On October 11, radioscopy distinctly showed a round shadow the size of a large orange at the right pulmonary base with an even opacity over its surface and two other shadows over the two pulmonary hilums, corresponding to enlarged tracheo-bronchial lymph-nodes. The upper limits of the large shadow were represented by a rather distinct line with its convexity uppermost. The lower limits could not be made out with precision as the opaque area blended with the hepatic shadow.

October 13.—Generalized pruriginous cutaneous eruptions composed of small urticaria elements.

In the night of October 14-15, there was another quite severe hemoptysis; injection of three centigrams of emetine. During the day there was some bloody sputum.

October 16.—Auscultation revealed a generalized bronchitis in both lungs. It is to be noted that the vesicular murmur was nowhere absent, but throughout the lower half of the right lung behind, there were pleural friction sounds and râles due to congestion. The usual medication for acute febrile bronchitis was prescribed—sodium benzoate, etc.—which resulted in abundant expectoration in which small grayish membranes characteristic of hydatids were detected. At this time it was no longer doubtful that, unless a copious vomit took place, the patient was about to empty the cyst and that perhaps the inflammatory reaction generalized throughout the bronchial tree indicated an irritative process due to contact with the cystic contents.

On account of the pyrexia, the respiratory reaction and also if possible to obtain a spontaneous cure of the cyst when once emptied, operation was postponed.

Up to October 21, the temperature remained high and the general health was mediocre, while until December 15, there were successive attacks of bronchitis with more or less profuse expectoration which upon several occasions contained hydatid membrane. But the general health declined and radioscopy showed the integral persistency of the cysts so that in order to cut short the oncoming cachexia operation was decided upon.

On December 15, an incision was made along the ninth rib about eight centimetres long, starting from a vertical line passing by the axis of the scapula. Resection of the rib and prudent incision of the pleura. No pneumo-thorax occurred. The parietal and visceral pleura were united by soft adhesions which were easily broken down with the finger. Some more important fibrous bands were divided with scissors in order to expose the lung cortex. Everywhere throughout the field of operation the pulmonary parenchyma was dark-wine color and everywhere the tissues could be depressed with the finger, so that it seemed difficult to expose the cystic pocket. However, a point was found in the depths where resistance was a little more marked.

An exploratory puncture with a fine needle was made at this point but the needle being plunged into different parts did not give issue to any fluid. A large calibre needle was then inserted and a drop of pus came away, but by aspiration hardly 3 c.c. could be withdrawn.

At this point an incision was made in the pulmonary parenchyma down to the collection—about one centimetre deep, which bled rather severely. In order to get better exposure the eighth intercostal space was incised at each end of the incision perpendicularly to its axis. After this it was easier to free the opening made in the cyst and draw its edges—upper and lower—out of the thoracic incision. The cavity was emptied of its pus, and the mother vesicle and numerous daughter vesicles filling it, an attempt was made to cleanse the interior of the cyst with ether, but the patient suddenly became suffocated and expelled blood from the nostrils and mouth. The narcosis

had to be stopped and the patient's condition, which was disquieting, improved by injections of caffein. The operation was completed by marsupialization and two large drains inserted into the cyst. There was little shock, salt solution and camphorated oil, 2 c.c., every three hours were all that was required.

December 16.—The temperature which had been about 100.8°F. on the preceding days rose to 101.2°F. in the morning and 103°F. in the evening. The wound was covered by a grayish membrane, fetid and freely oozing.

The temperature continued to range between 100.8°F. and 102.6°F., the aspect of the wound was disquieting, the odor was distinctly gangrenous and at the bottom of the wound there was a large blackish spot of slough. Gauze wet in Delbet's solution ( $\text{MgCl}_2$ ) was inserted around the drains.

December 29.—The wound was clearing up and the patch of slough was eliminated, still there was much oozing, the temperature remained up; on January 1, 1917, it was 103.1°F., and on January 10, 104°F. Nevertheless the cyst was becoming progressively more superficial and on January 12, the evolution became distinctly favorable. The temperature fell and rarely reached 100°F. in the evening.

On January 17 one drain was removed and on January 30 all drainage was stopped. Epidermization was complete on February 8 and the patient was discharged well on February 23, 1917.

The patient had a large sunken cicatrix which followed the movements of respiration. Auscultation revealed a bronchial souffle on the left and a little dullness near the hilum. A slight hemoptysis occurred on June 4, 1917, and it was feared that a small cyst in evolution might be present, but on June 12, radioscopy revealed an even transparency over both apices, a diffuse shadow corresponding to the operative area filling the costo-diaphragmatic sinus without any circumferential limits. On the left and right sides were shadows characteristic of tracheo-bronchial adenopathy. The liver shadow was everywhere homogenous.

CASE II.—*Large solitary suppurative hydatid cyst of the left pulmonary base coexisting with aortic disease. Hemoptyses. Diagnosis unrecognized in spite of radioscopy. Pneumotomy. Operative vomit. Recovery.*

Female, *æt.* 18 years, unmarried, entered hospital March 18, 1917, for hemoptysis. No distinct tuberculous heredity. Father and mother died when patient was young; one brother and two sisters in good health. For nine months the patient had had bronchitis with hemoptyses, the menses were suppressed, the amenorrhœa having lasted seven months and coincided with the frequently severe hemoptyses. She has emaciated and complains of effort dyspnœa.

On examination the paleness of the patient and a notable sudation were especially noted, emaciation was not very marked. The expectoration was rather free and hemoptoic.

Percussion over the left lung behind revealed dullness over the lower third of the lung. Over the right apex there was mild dullness with increased vibrations. On the left in the dull area the vibrations were diminished.

Auscultation revealed a decrease of the vesicular murmur in the dull area, with inspiratory râles over the entire left lung and fine râles in the left axilla.

Pulse tense and depressible, 100 per minute. Blood-pressure with Pachon's instrument: Maximum, 20; minimum, 9. Pulsating carotids, the aorta can be seen to rise in the suprasternal notch. The precardiac region receives an impulse at each systole. The heart's apex is in the sixth left intercostal space a little outwards and a thrill can be detected here. The cardiac dullness extends beyond the right edge of the sternum.

Over the aortic area and somewhat throughout the precardiac region auscultation revealed a double murmur which was not heard at the back. No capillary pulse, no crural double souffle.

The hemoptoic expectoration continued and the patient suffered from precardiac pain and distress. Arrhythmia.

October 13.—Positive Wassermann. Radioscopy: Cardiac shadow appears to be enlarged to the left with an expansive ectatic pouch on the aortic arch.

October 14.—A series of biniodide Hg. two centigrams, was begun and continued to October 30.

October 17.—Pain in left breast. The precardiac painful phenomena have subsided. Pulv. ipeca., 5 cg., 4 times daily for hemoptysis.

October 29.—Another hemoptysis. October 30, Hg. injections stopped.

October 31.—Some bloody sputum in the morning.

November 3.—Weight—92 pounds. Blood-pressure—maximum, 19; minimum, 8.

November 13.—Some palpitation. Ordered KBr, one gram daily.

November 18.—Some hemoptoic sputum.

November 30.—Weight—95 pounds.

December 6.—Treatment with Hg. recommenced.

December 11.—Precardiac paroxysms with small rapid pulse, but no dyspnoea.

December 12.—Pain in left hand and ring finger.

January 13.—Two precordial paroxysms of pain with persistent distress.

January 14.—Painful paroxysms.

January 17.—Weight, 94½ pounds.

January 20.—Severe hemoptysis.

February 14 and 17.—Hemoptysis.

February 19.—Severe hemoptysis; injection of emetine. To-day for the first time—excepting from December 13 to 17 when the evening temperature reached 100.6°F.—the temperature suddenly went up to 103.5°F. in the evening and in the evening of February 20, was 103°F. With a few rare remissions, the temperature curve remained between 100.6°F. and 102.5°F. until March 12, when it increased. February 25, weight—93 pounds.

March 13.—Morning temperature 102.5°F., evening 104°F. Signs of a fluid collection were detected over the entire left thorax, dullness in front reached to just under the clavicle. Traube's space distinctly resonant. Exploratory puncture gave exit to pus, but an attempt to empty the pus by aspiration failed.

March 15.—Radioscopy. The pulmonary clearness on the right contrasted with the shadow over the entire left pulmonary surface. Morning and evening temperature, 102.4°F.

March 16.—Temperature morning 103°F., evening 103.5°F. March 17, temperature, morning 103.5°F., evening 103.2°. Signs of fluid collection unchanged.

March 18.—Morning temperature 104.6°F. Extreme dyspnoea, lips violet. Exploratory puncture in eighth intercostal space withdrawing pus. The left thorax was arched with complete percussion dullness.

*Operation.*—Analgesia with sol. cocaine 1 per cent. Resection of tenth rib and incision of the pleura the entire length of the space left by resected rib. The pleural cavity was largely obliterated by loose adhesions easily broken down with the finger. All the walls were flexible and yielding excepting above where a hard mass was detected, bounding with each systolic impulse, but without any expansion.

It was impossible to split open from below upwards, the resisting walls of this tense pouch evidently containing a fluid collection. The pleural cavity was obliterated by contact of the collection with thoracic walls.

The patient, lying on the healthy side, became more and more dyspnoic. The local analgesia was not enough for a more extensive operation and general narcosis was too risky. An exploratory puncture above the ninth rib gave exit to pus. The upper edge of the incision was freed upwards by a stroke of the knife, and the eighth intercostal space was freely incised until the pus was reached. When this incision was made the patient had a paroxysm of suffocation with an abundant bloody purulent vomit, while at the same time a large gelatiniform, putrid hydatid membrane, dirty gray in color, fell into the hands of the surgeon who was busy sponging the pus flowing from the incision with each spell of coughing. Injection of morphine, 1 centigram; caffeine, 25 centigrams.

The operation was quickly completed by inserting two drains in the incision in the eighth intercostal space and another at the declivous part coming out below the ninth rib through the first incision. Patient put to bed in sitting position.

After the operation the evolution was apyretic and perfectly regular and she was discharged cured thirty-five days later.

June 11, 1917.—Clinical examination showed the patient in better health than she had ever had. Weight, 98 pounds. She is being given an intensive Hg. treatment, daily injections of Hg. bini-odide, 2 centigrams.

The operative cicatrix is remarkably flexible, with the shape of an elongated "S" and then curves behind upwards.

*Percussion and Auscultation.*—*In front* resonance about equal over both apices. There is perhaps a slight decrease of resonance over the right apex where the vibrations appear to be somewhat increased. Vesicular murmur equal on both sides. *Behind* over the entire left lung slight dullness, vibrations equal. Evident decrease of the vesicular murmur. At the lower half of the left thorax the duration of the expiration is equal to that of inspiration.

The stethoscopic sounds of the cardiovascular system exactly coincide with those previously mentioned.

June 12.—*Radioscopy.*—Both in front and behind the clear pulmonary areas are equal. The costo-diaphragmatic sinus on the left side is remarkably free. No tracheo-bronchial adenopathy. The aortic disease is evident. The aortic shadow extends one finger's breadth beyond the left sternal edge; pulsation and expansion of the aneurysmal pouch are distinct. On the edge which on the left limits the projected shadow, is another darker shadow about the size of a finger-nail, having about the opacity of bone and which, when the projection is changed, appears to be imbedded in the aortic wall and follows the movements of expansion. No cyst in the liver could be discovered.

The patient remains an aortic subject, but she is completely cured of her pulmonary hydatid cyst.

CASE III.—*Solitary, non-suppurating hydatid cyst of the middle lobe of right lung. Clinical diagnosis confirmed by radioscopy. Pneumotomy. Recovery (see Fig. 1).*

Female, *æt.* 21 years, unmarried, entered hospital on March 27, 1917, service of Doctor Pélissier, who had seen the patient in consultation on March 25, because of "spitting of blood for several months" and emaciation which continued to progress in spite of injections of Na cacodylate, she having been suspected of having pulmonary tuberculosis.

The history of the case is briefly that, for the first five months, the patient had a painful point in the right thorax varying in site, and pain in the right shoulder. For about three months the patient has complained of some effort dyspnoea, but she continued her work.

FIG. 1.



FIG. 2.



FIG. 3.





Suddenly bright red severe hemoptyses occurred with paroxysms of coughing. There was some fever but the temperature was not taken at the time.

The patient was a large, thin girl, with a yellow tint and looked as if she were suffering. Examination of the chest revealed normal resonance behind and in front throughout the left thorax, the cardiac area being excepted. On the right there was dullness in the lower two thirds of the lung both in front and behind, with an almost normal resonance at the base.

Auscultation, normal on the left, revealed an area of respiratory obscurity in the mid portion different in front and behind. In front, the vesicular murmur was completely abolished in the two lower thirds of the lung; it could just be heard under the clavicle with a rougher tone than on the left. Below, the lung appeared to be normally permeable without superadded râles. Absolute respiratory integrity at the base.

The vocal vibrations could be heard very well behind, but in front they were distinctly diminished on the right side throughout the entire mid-area. There were no pleuritic signs, egophony or aphorous pectoriloquy.

Given this symptomatology which did not fit very well with the previous diagnosis of pulmonary tuberculosis a search was made for an encysted intrathoracic collection, interlobar pleurisy or a hydatid cyst of the lung. By close questioning, the patient said that the bloody expectoration was occasionally accompanied by transparent pellicles, "like grape-skins."

Radioscopy revealed an admirable picture of an hydatid cyst of the right lung (see Fig. 1). In the midst of the clearness of the lung tissue normally preserved at the apex and base, a dark disc was seen with an almost geometrically circular contour. The somewhat grayish shadow is a little darker at the centre; the area appears with greater distinctness when the patient faces the screen with the ampoule at the back. In oblique or lateral positions the image appears to be nearer the anterior than to the posterior part of the thorax, as might be expected. The projection of the image on the posterior wall was as follows: The lower limit of the cystic shadow extended

slightly below the lower edge of the eighth rib, and above, a little beyond the upper edge of the fourth rib.

Weinberg's sero-reaction was negative; eosinophilia was not looked for. On the other hand, the patient informed us that she owned no dog, but frequently ate water-cress.

*Operation* (April 2, 1917).—General ether narcosis. Operator, Doctor Pélissier.

The shortest approach to the cyst was certainly by the anterior route, but in order to obtain better drainage it was preferred to approach the cyst by the postero-external route, with the result that some operative difficulties ensued, as I shall show.

Resection of the eighth rib was done through an incision about twelve centimetres long, starting at two fingers' breadth from a vertical line drawn down from the tip of the scapula. The pleura was then opened. It was found covered with loose adhesions which were easily broken down with the finger.

Above and in front, a little above the healthy and normally elastic pulmonary parenchyma could be felt the renitent cyst. Forced retraction of the ribs with Tuffier's retractor did not give sufficient access to the cyst, which, undoubtedly adherent to the anterior wall, could not be drawn up to the incision. Consequently, a second incision four centimetres long, was made at the anterior end of the first incision and perpendicular to it which resulted in a flap which when thrown back allowed one to resect seven or eight centimetres of the seventh rib. The cyst was then perfectly accessible but nevertheless it could not be drawn up.

The pleural cavity was therefore carefully packed with gauze strips and aspiration of the cyst with Potain's apparatus was done. The fluid withdrawn was characteristically limpid. The cyst was then dried and its adventitia incised, the cyst membrane was removed but did not contain any daughter vesicles. The resulting cavity was swabbed out with ether which gave rise to coughing but no tendency to suffocation, while at the same time a little bloody froth came away from the patient's mouth, indicating a communication with the bronchial tubes.

For this reason one did not dare to reduce the cyst without drainage and in spite of the depth of the pocket an attempt was made to

stitch it to the borders of the skin incision which was successful. Two large drains were inserted into the cystic cavity and the pleura was loosely packed with gauze. Patient was returned to bed with a rather soft pulse at 90. Salt solution, 500 c.c., and an injection of 2 c.c. of camphorated oil were all that was necessary.

For the first four days after the operation everything went well; the temperature did not go above 100°F. and on April 6 it was 99°F. in the evening. Daily change of dressings moistened with Delbet's magnesium chloride solution (12 grams per 10,000 c.c.) was done. Then the wound became covered by an ill-smelling grayish coat, which cleared up and left a healthy bright red surface.

However, the patient's state changed on April 7. There was some oppression, the morning temperature was 101.2°F., in the evening 102°F. On the following days signs of right pulmonary congestion developed although the extreme temperatures were 101.5°F. and 104.3°F.

April 15.—A severe hemorrhage suddenly developed in the wound, soaking the dressings and bed, the pulse became small at 152 beats. At the same time an intense hemoptysis occurred in the midst of paroxysms of coughing. Morphine was given subcutaneously for the cough, while 8 centigrams of emetine and 25 centigrams of caffeine and 500 c.c. salt solution were given for the acute anæmia resulting. Camphorated oil was injected every three hours. The hemorrhage stopped in the evening. Temperature 105°F., respiration perfectly calm. Dressings changed, no oozing.

April 16.—Morning temperature 98.6°F., evening 100.8°F. This post-hemorrhagic defervescence should be noted.

April 17.—Evening temperature 104.3°F., pulse 100. General condition satisfactory. Injections of camphorated oil continued.

After this the temperature tended to go down and on April 26, it reached 98.8°F. in the morning; evening 100.6°F.

May 8.—Temperature normal. The secretion in the wound was lessening, the drains had been progressively shortened and smaller ones introduced. All drainage was stopped on May 22. The wound was almost completely healed in the first days of June.

June 12, 1917.—Epidermization of the wound, rather superficial, was not perfectly complete. There was no fistulous tract. By

auscultation nothing was noted excepting a decrease of the vesicular murmur in the area of the operation. The general health was remarkably good. Patient was visibly fattening.

July 2, 1917.—Radioscopy showed an integral clearness in both apices and only the area of operation in the middle lobe was the seat of a slight diffuse shadow, but it was impossible to discover the slightest evidence of pleural adhesions.

The liver shadow was normal without any opaque spot that might lead one to suspect a cyst.

CASE IV.—*Suppurating hydatid cyst in the base of right lung, discovered a few months after hepatotomy for a cyst of the liver. Hemoptysis. Diagnosis made by the patient's history, clinical examination and radioscopy. Pneumotomy. Recovery.*

Female, æt. 20 years, single, entered hospital April 10, 1917.

May 9, 1916.—Patient had been treated for two years for tuberculosis of both apices. In reality, there exists a manifest prolonged expiration, some sibilant râles and some dullness with exaggerated vibrations, particularly over the right apex.

For some time the patient had been dyspnoëic and unable to do any exercise. There are signs of right-sided pleurisy with a large fluid collection. Thoracentesis on May 11, withdrew 850 c.c. of limpid liquid resulting in the immediate diagnosis of hydatid cyst of the liver with pseudo-pleuritic symptoms.

This diagnosis was confirmed on the following day by the development of a generalized eruption of urticaria with a temperature of 102.2°F. An attentive examination showed that the liver was enlarged with signs of a voluminous liquid tumor. On May 19, 1916, Doctor Pélissier removed an enormous non-suppurating hydatid cyst containing 1500 c.c. of limpid fluid but without any daughter vesicles.

Recovery normal, the patient returning to flourishing health and seemed to be definitely cured for the lapse of several months. However, towards the end of 1916, the health began to decline.

March 25, 1917.—Patient underwent a complete examination because a few days before she had had severe hemoptyses which caused her considerable alarm.

There was dullness at the base of the right lung behind, with a slight dullness over the apex. Vibrations existed throughout the

right lung and were increased at the apex. The vesicular murmur could be distinctly heard at the apex. Expiration, whistling. Respiratory obscurity at the base.

As radioscopy was considered necessary, before going to hospital, the patient was treated at home for *extraordinarily severe and repeated hemoptyses* which did not cease by rest and injections of emetine.

Entered hospital on April 10. Radioscopy revealed the characteristic spots of bilateral tracheo-bronchial adenopathy and integrity of both apices. Attention was directed to the right pulmonary base where at two fingers' breadth above the diaphragm a rounded shadow the size of a small orange could be seen (see Fig. 2). Its centre was opaque, its contours ragged. Weinberg's reaction positive. Morning temperature 99°F., evening 101°F.

Operation on April 18, by Doctor Péliissier. General ether narcosis. The cyst appeared to be distinctly more accessible by the posterior approach. Incision over the eighth rib eight centimetres long and beginning at the axis of the scapula. Resection of eight centimetres of rib; puncture of the pleura to let air in slowly. The pleura did not appear to be free.

It was found to be divided off into partitions by loose adhesions so that only a limited pneumo-thorax resulted.

The rest of the operation was one of ideal simplicity. The cyst was at once recognized by its white cartilaginous aspect against the wine-red pulmonary parenchyma. The pleura being protected with gauze the cyst was punctured with a fine needle, giving exit to a recently infected cloudy liquid. A large needle was then inserted, but as the fluid did not come away readily the adventitia was incised. With gauze mounted on forceps slightly moistened with ether, the cyst cavity was cleansed and the mother membrane, which did not contain any daughter vesicles, was removed. Although collapsed, it was larger than the radiography would have led one to suppose, it being much larger than an orange. The borders of the incision in the adventitia were easily brought up and sutured to the skin incision and the cyst could be correctly marsupialized. Two large drains in cyst cavity.

At the end of the operation there was some pink froth on the lips

and in the nostrils; pulse small and rapid. Temperature at 6 p.m., 99°F. Injected 2 c.c. camphorated oil, 500 c.c. salt solution and 8 centigrams of emetine as a prophylaxis for hemorrhage.

April 19.—General condition excellent. However, the wound was covered with a slightly ill-odorant grayish coating—injection from the bronchial tubes. Delbet's magnesium chloride dressing. Temperature, morning 101°F., evening 101.5°F. Injection of camphorated oil and 500 c.c. salt solution.

April 20.—Magnesium dressing. Temperature 101.3°F., evening 101.5°F. Camphorated oil 2 c.c.

April 21.—Wound clearing up, bright pink color. After April 22, the dressings were changed less often, the temperature remained around 100.5°F. and on May 10 it was normal. The drains were removed on May 16.

July 2, 1917.—General health excellent. Patient has fattened. Operative cicatrix slightly depressed and presents a tiny fistula.

Examination of the thorax revealed a very slight dullness in front over the right apex, with slightly increased vibrations and prolonged expiration. The sonority, vibrations and vesicular murmur offer nothing abnormal throughout both lungs in front and behind.

Radioscopic examination showed bilateral tracheo-bronchial adenopathy quite as marked on the right as on the left. No lesion radioscopically visible in the right apex although it was clinically suspected of tuberculosis.

At the base of the operated lung the transparency was not quite as perfect as on the other side, but the difference was very trifling. There was no apparent cyst in the liver and all traces of the large cyst, removed in 1916, had completely disappeared.

CASE V.—*Non-suppurating hydatid cyst of the base of the right lung. Limpid hydatid vomit. Operation refused (see Fig. 3).*

Male, æt. 10 years, entered hospital December 14, 1916. On October 30, 1916, the child, in apparently excellent health, was seized by severe vomiting of bright red aerated blood, followed by the expectoration of thick greenish sputum with a fearful odor.

The child was seen on November 3, 1916. He coughed, expectorated bloody sputum, but there was no temperature and he was not exhausted. Auscultation revealed bronchial respiration.

Another and more serious hemoptysis frightened the family and the child was brought to hospital on December 14, 1916.

The mother had died in 1915 from post-partum complications following the birth of a sixth dead-born child. Four brothers and sisters of the patient are in good health.

Born at term the patient developed normally. Typhoid fever four years ago.

In spite of the severe hemoptysis and persistence of bloody sputum the general health was not influenced. There was slight dullness over the left apex with some pain on pressure. At this point the expiration was whistling and some fine crackling râles on inspiration. Bronchial respiration, no bronchophonia. Heart-sounds normal.

The child continued to cough but did not have another hemoptysis. In front over the left apex there was slight dullness, whistling inspiration and rough regular expiration.

The temperature, which during this short period varied between 98.6°F. and 99.5°F., reached 101°F. on the evening of December 22.

Cutireaction with tuberculin made on December 18, was negative.

December 24.—Radioscopic examination showed normal clearness over the apices and an oval shadow at the right base, the costo-diaphragmatic sinus remaining free. Weinberg's reaction negative.

January 3, 1917.—For the past two or three days the child did not look well. He coughed more and complained of pain in the right thorax; sputum usually bloody. Temperature, morning 99.6°F.; evening, 103°F.

January 4.—The leucocytic equilibrium did not indicate eosinophilia. The formula was: Polynuclears, 74; lymphocytes, 23; large mononuclears, 3; eosinophiles, 0.

Morning temperature, 99.1°F.; evening, 100.5°F. A paroxysmal cough expelled hemoptoic sputum. Percussion revealed slight dullness over right base.

January 6.—Morning temperature, 100°F.; evening, 103.2°F. A second radioscopic examination gave an absolute obscurity at the right base. The upper limits did not become displaced when the child was lying on the side; costo-diaphragmatic sinus was opaque.

January 7.—Dry, paroxysmal cough. Hemoptoic sputum. Per-

cussion still gives slight dullness over right base, but auscultation showed that the vesicular murmur was hardly diminished, as well as the presence of fine inspiratory râles. No signs of a fluid collection. Temperature lower.

January 10.—Puncture was made in the right pulmonary base withdrawing an almost clear *yellow fluid*, containing some red blood-corpuscles, a few lymphocytes and numerous polynuclears. The white cell count of the blood gave a normal amount, *viz.*, 5200.

January 12.—Condition stationary.

January 14.—Expectoration of a *limpid fluid*, like “spring-water,” without membranes, or vesicles, followed by vomiting and a very severe hemoptysis. Morning temperature, 98.9°F.; evening, 103.1°F. Emetine 3 centigrams subcutaneously.

January 15.—Morning temperature, 98.6°F. Another hemoptysis. Evening temperature, 102.2°F.

January 17.—Dullness, friction sounds and fine râles over the right base. Morning temperature, 100°F.; evening, 100.5°F.

January 21.—Cough continues, absolute dullness over right base, with respiratory obscurity and decrease of vibrations. No râles, no bronchophony.

In spite of the amelioration of the general condition dullness still persisted, but the vesicular murmur was progressively becoming more distinct. There was still some friction sounds and fine râles at the right base. On January 23, the evening temperature was 101.2°F., but never again went above and for a long period—from January 27 to March 26—the temperature was never higher than 100.5°F. The subjective symptoms—dullness, fine râles and friction sounds—continued to be present.

April 3.—An eruption of urticaria developed but the temperature did not go up.

April 7.—The erythematous patches have disappeared.

April 21.—Radioscopy still shows a round shadow at the right base.

June 3.—General health has remained good. The same symptoms exist at the right base. Wassermann positive. A surgical interference was suggested to the patient's family, but was refused.

June 23.—General health good. Weight—59 pounds. Appe-

tite good. The mucosæ are a little pale. Always some cough. Patient never complains of any spontaneous pain in thorax. No hemoptysis. Thorax does not bulge. Percussion sound normal over both lungs excepting at right base. Auscultation revealed some mucous râles scattered throughout the right lung. Respiratory obscurity at the right base, and above this area the respiration was somewhat whistling. At the lower part there were some inspiratory râles, while the vibrations were normal excepting in the dull area where they were diminished. No cardiac lesion.

*Radioscopy.*—With the patient's back facing the ampulla of the instrument two series of chains of small lymph-nodes to the right and left of the spine reveal a bilateral tracheo-bronchial adenopathy. The left lung was uniformly clear. On the right, almost in contact with the diaphragm a circular shadow was seen, with quite distinct contours, and in extent was that of a one dollar silver piece.

#### ETIOLOGY

I have new data to offer to the description of the *tenia echinococcus*, the causal agent of hydatid cysts. In referring briefly to the etiology I shall here consider the frequency. Hydatid cysts in general are not especially uncommon in Algeria. In Algiers, the frequency of hydatid cysts of the lung as compared with other surgical affections and the frequency of pulmonary localization as compared with other localizations of the echinococcus is what I shall discuss, employing for this purpose the records of the Clinic for Surgical Diseases of Children and the records of the General Surgical Clinic (male wards).

*Frequency as Compared with Other Surgical Affections.*—In children from two to fifteen years of age, Alberti found out of a total of 9000 cases admitted to the Clinic in the space of twelve years that there were only three cases of pulmonary hydatids. Alberti's statistics stop in July, 1911.

From July 1, 1911, to June 20, 1917, 2788 patients were received into the Children's Clinic for various acute and chronic affections requiring surgical treatment. Of this total of 2788 cases only two were hydatid cysts of the lungs so that adding Alberti's figures to mine we find that there were five cases out of a total of 11,788 patients. Hence the frequency of hydatid cysts of the lung encoun-

tered at the Children's Surgical Clinic is one cyst out of every 2357 cases admitted.

*Frequency in Adult Males (from 16 years upwards).*—From January 1, 1903, to June 21, 1917, hence fifteen years, the male surgical service of the Mustapha Hospital (service of Professor Vincent) admitted 5452 patients. During the year 1915, the ward was used for military surgery, so that the patients treated during this period are naturally excluded from the total number admitted. Of these 5452 cases there were 13 pulmonary hydatid cysts, otherwise one cyst out of every 419 cases.

It is remarkable that there is such a difference in the ratio of frequency between a surgical service of adult males and one of children—boys and girls—namely, 1:419 and 1:2357 respectively.

But these figures are based on sufficiently important statistics so that there can be no question of mere chance. For example, in 1913, Professor Vincent met with four pulmonary cysts out of a total of 534 patients, and two out of 475, in 1914.

Statistics which would only comprise these two consecutive years would lead to an erroneous appreciation of the frequency of hydatid cysts of the lung. In statistics covering fifteen years this concentrated percentage becomes diluted, the years in which quite a few cases were met with being balanced by years of great infrequency—no case being met with in 1904 to 1908 and 1910 to 1915—so that the average comes nearer to the reality.

But the figures I have given were obtained in children and adult males. Thanks to Doctor Pélissier, I am able to offer total statistics somewhat more reduced, but in which the age and sex are confounded so that they better express the frequency of hydatid cysts of the lung for a given number of patients coming under the care of one surgeon. In several hospital services—including a children's clinic—and in private practice Pélissier, out of a total of 3728 operations, encountered cysts of the lung four times. Hence, this surgeon, at Algiers, averaged one cyst of the lung out of 932 patients.

Now, of these 3728 patients, 2404 were children under fifteen years of age and only one case of hydatid cyst of the lung was met with; 1324 patients were over fifteen years old and there were three

pulmonary cysts observed, otherwise, one cyst out of 441 patients. Hence, comparing these figures with those I have given, we find:

In children .....	1 in 2357	1 in 2404
In adults .....	1 in 419	1 in 441

The coincidence is striking.

It may be said that this represents only the approximate truth because my investigations were only made in surgical cases, but I will let the facts speak for themselves:

Hydatid cysts of the lung—at least in Algiers—bring the patient to the surgeon much less frequently than other so-called surgical affections. Surgeons specializing in children's diseases meet with one pulmonary cyst out of 2300 to 2400 patients. General surgeons meet with this lesion once out of every 400 to 450 patients.

Undoubtedly a great number of these cysts exist but do not come under the care of surgeons, especially those cases in which the cyst empties itself by a vomit and then spontaneously recovers.

Dévé appears to believe that the majority of these cases never reach the surgeon. The physicians of the region gave Dévé their complete statistics, each covering at least several years' practice. Nineteen of the cases thus collected ended in a vomit with spontaneous recovery, the patients remaining permanently cured after a number of years. But Lepicard<sup>1</sup> states that this was a fortunate series.

My figures show that: (1) at the Mustapha Hospital in Algiers, hydatid cysts of the lung are relatively rare in the surgical services, and (2) that this lesion is far less frequent in children than in adults.

I was unfortunately unable to collect the statistics from the medical services, but I am inclined to believe that the figures I have given express the relative rarity of cysts of the lung in medical practice in Algiers. Those physicians with whom I have conversed on the subject said that they had not met with many cases and those that they did meet went to the surgeon.

It is a fact that the lesion under consideration is uncommon in children and various hypothetical explanations can be offered, such as tardy infestation in late childhood, or the slow development of the vesicular parasite only giving clinical manifestations at a

<sup>1</sup>“La vomique hydatique pulmonaire; sa valeur curative,” *Thesis*, Paris, 1912.  
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rather advanced age of the patient—after the age of fifteen years. It seems to me useless to develop these considerations which have no clinical or experimental foundations.

The following table shows the comparative frequency of the localizations of hydatid cysts in Algiers:

	Dr. Lemaire	Dr. Alberti	Prof. Vincent	Dr. Péliassier	Total
Liver .....	70	18	47	9	144
Lung .....	14	2	13	4	33
Other viscera	25	5		3	33
Total .....	109	25	60	16	

In the adult there consequently are thirteen cases of pulmonary hydatids for forty-seven in the liver, otherwise about one pulmonary cyst for four hepatic cysts.

Cadi, in his "*Etude de l'échinococcose en Algérie*,"<sup>2</sup> publishes the statistics of echinococcosis treated at the Mustapha Hospital from 1898 to 1913. He found 144 cases of liver hydatids and 19 of the lung or pleura. Otherwise put, one pulmonary cyst for every 7.5 cases of liver cyst. But I question if Doctor Cadi has not made some omissions, because for example, for the year 1913 he mentions only one case, while in reality Professor Vincent operated that year on four.

From the table above given the result is one cyst of the lung for 4.3 of the liver and this ratio is not very different from what is found in other countries. Lemaire's statistics comprising a total of 2169 cases of hydatids in various parts of the body—derived from Davaine, Cobbold, Finsen, Neisser and Davies-Thomas—show 1119 cysts of the liver as against 206 in the lungs, otherwise put, one pulmonary cyst for every 5.4 in the liver.

Prat, in his statistics compiled in Uruguay in 1913<sup>3</sup> has collected 471 cases and he gives the proportion of 70 per cent. for hydatids of the liver, and 20 per cent. of those of the lung, otherwise, one pulmonary cyst for every 3.5 of the liver.

Finsen's statistics, quoted by Lemaire, show that in Iceland there are seven pulmonary cysts for every 256 other sites, that is to say, one cyst of the lung for every 36.5 cysts elsewhere in the body. This ratio is very low compared with the figures I have given.

<sup>2</sup> *Thesis*, Algiers, 1915.

<sup>3</sup> "Revista de los Hospitales," Tome vi, no. 8, 1913.

According to Bird, who believed that infestation might take place in the respiratory tract from inhalation, the rarity of these cysts in Iceland is due to the prevailing damp climate. Dryness favors the pulverization of dogs' excreta which will be inhaled with the dust.

As to the frequency according to sex admitted by the majority of writers as being equal, I will merely offer the three following statistics:

	Females	Males	
Lemaire .....	10	8	= 18
Alberti .....	0	2	= 2
Personel .....	4	1	= 5

What is striking in the Algiers' statistics is the extreme infrequency of echinococcosis among the indigenous Mussulmen. Out of the 26 cases collected by Alberti there was only one young Arab, *æt.* 5 years, who presented an hydatid cyst in the subcutaneous cellular tissue.

The echinococcus is also occasionally met with in adult Arabs, in the liver or other viscera or tissue, but pulmonary hydatids are exceptional in them. In Cadi's nineteen cases of pulmonary hydatid cysts no Mussulman appears upon the list, while in a total of 38 cases reported by Lemaire, Alberti, Vincent and myself there was no indigenous subject among them.

I am aware of three other cases in Algiers, but the details were not sufficiently complete to find place in my statistics. One was an adult Israelite and two reformed soldiers, one an Italian, the other a Frenchman, all three with pulmonary hydatid cysts. Abadie, of Oran, is the only observer in Algeria who has reported a case of primary hydatid cyst of the lung in an indigenous Mussulman. The patient was *æt.* 27 years.

It appears difficult to explain this apparent immunity among the Arabs. Lemaire attributed it to the alimentary régime of Mussulmen or to the perhaps rarer infestation of the Kabyle dog.

The Arab especially eats mutton, very little beef and no pork at all as it is forbidden by the Koran. Now, at the slaughter-house in Algiers, 1.03 per cent. of the ovines, 7.55 per cent. of the porcines and 9.88 per cent. of the bovines killed are infested by the echinococcus. Still the Arab eats meat rarely, five or six times a year at the very most. On fête days—there are only two—and whenever he

invites a distinguished foreigner to his house, meat is served. Hence Arabs and Kabyle dogs are less exposed to infestation than are Europeans and their dogs.

#### PATHOGENIC INTERPRETATION

My five cases consist of four single primary hydatid cysts of the lung with perfect integrity of all the other viscera, especially the liver, and a pulmonary cyst evolving after a large primary cyst of the liver.

Before taking up the pathogenic problem raised by isolated infestation of the lung by the echinococcus, I must first discuss the pathogenic interpretation of Case IV.

When two cysts coincide in the same patient, but without contemporary clinical manifestations, the tendency is to admit that the cyst which first developed is the initial cyst and that the other is secondary and should be attributed to a graft. I believe that the two cysts in Case IV were independent of each other and resulted from a double infection. My arguments are as follows: The first cyst operated on was the large one in the liver. It was a closed cyst with a limpid content, hence its spontaneous evolution could not have given rise to a graft.

It might be thought that a scolex at the time of the puncture made a few days before operation—pyrexia and urticaria—or at the time of the interference, in spite of the precautions taken—cleansing the pocket for four minutes with a 1 per cent. formol solution—might have been transported by the blood or lymphatics to the lung.

If it be admitted that the graft occurred during puncture or operation, it must also be admitted that the cyst developed with extraordinary rapidity, because ten months after the hepatotomy, pneumotomy resulted in the extraction of a cyst the size of a fetal head.

It is known that the duration of normal evolution of an hydatid cyst varies with its size. At the end of five months in the subserous hepatic parenchyma the vesicle does not measure more than one centimetre in diameter (Brumpt).

On the contrary, in the lung, on account of the weak resistance of the pulmonary parenchyma offered to the development of the cyst, its evolution will be relatively more rapid. According to Escudero<sup>4</sup>

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<sup>4</sup> "Argentina Medica," vol. vii, 1909.

two years elapse between the infestation and the development of the clinical phenomena.

It therefore seems to me unlikely that, in the case under consideration, such an important growth could have taken place in so short a time and I believe that the hepatic and pulmonary cysts were each primary, although I in no way infer anything in respect to their ages. I would add that before the hepatotomy the diagnosis was so certain that radioscopy was not necessary and this is perhaps why the pulmonary cyst, which at this time was small, was not diagnosed.

But in the other patients—Cases I, II, III and V—the solitary pulmonary cyst in each was unquestionably primary. By what route did the scolex reach the lung? This question cannot be answered positively and I can only mention those possibilities admitted by some and to insist upon those that I believe to be the most likely.

To penetrate the human organism from without the hexacanthous embryo may follow either the digestive or respiratory routes.

(A) *The Digestive Route.*—The ovum, dissolved in the stomach by the gastric juice, is freed and with its hooklets it emigrates through the walls of the intestine. Dévé fed a squirrel with segments of tenia and two days later found numerous cysts in the lungs and pleura.

From the intestine the parasite may reach the lung by way of the circulation or the lymphatics.

(a) *The circulation* offers two possibilities; the direct route and the transhepatic route. The most likely hypothesis, because it is the simplest, is that of the direct route which follows the great circulation to the right heart and then the pulmonary artery. By injecting an emulsion of scolexes into the vein of a rabbit's ear Dévé was able to produce a pulmonary echinococcosis.

This theory allows one to admit two different vascular routes followed from the intestine. According to Dévé, it is possible for the embryo to perforate the duodenum whose posterior wall is deprived of peritoneum, hence it can migrate into the system of tributary portal veins of Retzius—hepatic localization—or to the inferior vena cava and from here by the intermediary of the lesser circulation, it can be transported to the lung.

Chachereau believes that the embryo passing into the inferior

hemorrhoidal veins, affluents of the inferior vena cava, may likewise be drawn into the lesser circulation.

*The transhepatic circulatory route*, admitted by Behr as possible, supposes a more complex mechanism. Since the embryo measures from 20 to 25 $\mu$  (Brumpt) or from 30 to 35 $\mu$  (Castaigne) it can penetrate the portal-hepatic veinules, it traverses the very fine capillary network of the liver, moulding itself like a red blood-cell in its progress and by the suprahepatic veins reaches the vena cava and thence the right heart.

(b) *The Lymphatic Route*.—Since Dévé's experiments in which this observer noted the development of primary cysts in the mediastinal lymph-nodes, the theory put forward by Neisser of emigration by way of the lymphatics assumes greater importance. The parasite reaches the thoracic duct from the chyliferous ducts, from here to the left subclavian, the superior vena cava and the pulmonary circulation.

(B) *The Respiratory Route*.—Until recently Bird's opinion of infestation by way of the respiratory system was considered inadmissible on account of the impossibility of the bronchial mucus to liberate the embryo from its envelope, but at present it has been again accepted.

Lemaire, in 1903, already drew attention to the possible part played by the white blood-corpuscles in relation to the embryo. By destroying the envelope of the ovum phagocytosis may liberate the parasite.

Now, in 1907, Dévé showed that the action of the digestive juices was not necessary for putting the embryo at liberty. In reality, the subcutaneous injection of an emulsion of *tenia's ovi* will give rise to a local development of cysts.

The fact that out of the five cases I have met with there was only one of double injection of the liver and lung and in the other four there was a primary solitary cyst of the lung without any localization in the liver—controlled by repeated radioscopy—led me to willingly accept the direct blood route or the respiratory system as the route followed by the parasite to reach the lung.

I have vainly searched for the experimental proof or demonstrative data in recent literature which would permit one to assign

infestation of the lung by the echinococcus to a simple pathogenic mode.

#### PATHOLOGY

The data obtained in my cases drew my attention to various points in the pathology of varying importance and to which I shall refer.

It is an established fact that hydatid cysts of the lung are generally solitary. In this respect Davaine said that "it was easier to find a cyst in each lung than two cysts in one lung." Out of a total of 83 autopsies, Hearn found ten cases of multiple cysts, but it is evident that these are the ones that will probably end in death of the subject.

From the viewpoint of the site of the pulmonary cyst, I find the following peculiarities in my cases: In three the cyst was in the lower lobe on the right, once in the left lower lobe and once in the middle lobe of the right lung. Thus the classic notion of the frequency of the cyst in the right pulmonary base seems to be born out, but an absolute conclusion cannot be drawn.

In Lepicard's fourteen cases, in ten the cyst was in the right lung, in four in the left, seven times in the apex, six times at the base and once in the middle lobe.

The depth of the cyst in the pulmonary parenchyma is still more interesting. Three of the cysts I observed were in almost immediate contact with the pleura (Cases II, III, IV). In Case V, where no operation was allowed, it certainly seemed as if the process was a parapleural cyst, given the pleural reaction present and the data obtained by radiography.

The cyst in Case I was more deeply seated, there being about a centimetre's thickness of pulmonary parenchyma interposed between the hydatid and pleura.

It is perhaps to this superficial localization that the constant pleural reaction noted in my cases was due. In one of the cases (Case II) the anatomical situation of the cyst was such that it might at first sight have been mistaken for an hydatid of the pleura.

The subject of primary hydatid cysts of the pleura has given rise to much discussion, but Dévé, by his experimental work, has shown that this lesion is unquestionably a possibility. It had previously

been shown that the embryo of the parasite when it has reached the pleura is covered by the endothelium, thus becoming subserous, may become pedunculated and fall into the pleural cavity, surrounded by fibrous tissue.

In practice it is difficult, not to say impossible, to distinguish a voluminous cyst of the pulmonary cortex from one developing in the pleural cavity. Professor Vincent and Doctor Vincent have presented new data on this subject of pathology. In two of their cases they found a non-suppurating hydatid covered by yellowish membrane and bathed in a cloudy serous fluid. In a later phase these membranes became organized into fibrous adhesions with the development of a pachypleuritis. It is evident that when suppuration exists it is largely the result of an inflammatory process, but these observers have shown that pleural reaction may exist without suppuration of the cyst.

It is difficult to say if the reaction is due to mechanical or toxic irritation of the serosa, but I can say that in Cases III and V there was a pleural reaction although the cysts were intrapulmonary and there was no suppuration. Case V is a fine example of pleurisy as a secondary process. Experimentally, Dévé has produced a secondary pleurisy in the monkey and Faisans observed a case in which there was a pleurisy with marked eosinophilia. Eosinophilia was not looked for in the exudate withdrawn by puncture, but the clinical circumstances and discordancy between the polynucleosis and normal leucocytic count in the blood would seem to indicate that the cyst was directly or indirectly the cause of the pleural collection.

The peripheral situation of the cyst brings up another question. Lepicard points out that inversely from central cysts, those in the pulmonary cortex only open into the bronchial tubes at a late date in their evolution after they have become very large. My four cases operated on were very large, but as they all were in the cortex only two ruptured and were emptied by a vomit. Case I, a parapleural cyst, gave rise to repeated fractional vomits; in Case II, when the cyst was incised, a purulent and bloody vomit occurred; Case V, which was not operated had a limpid vomit and radiosopic examination showed that the cyst was in the pulmonary cortex and had become reduced in size.

The question now arises as to whether a pulmonary hydatid possesses an adventitia as that present in hydatids of the liver. It is generally believed to exist, but its thickness and make-up have been much discussed. The thickness must vary from one case to another, likewise the histology of the membrane. Escudero supposes that in central cysts the pulmonary alveolæ around the cyst become compressed as the hydatid increases in size. The atelectasic alveolæ hence form a rather thin adventitia around the cyst. The atelectasis ceases as soon as the cause of the compression is removed, that is to say, when rupture has occurred. The cystic cavity has a tendency to rapidly become obliterated on account of the elasticity of the pulmonary parenchyma.

On the contrary, in cortical cysts the hydatid provokes a permanent sclerosis and in these circumstances the pericystic membrane is thick and incapable of decreasing or collapsing so as to occlude the cavity left by the cyst after rupture or excision. To the sclerosis pleural adhesions become superadded and if infection ensues the resulting pericystic inflammation still more increases the thickness of the adventitia.

In three of my operated cases a thin, soft adventitia was found which collapsed when the cyst was emptied. In Case II, in which suppuration had developed, the adventitia was very thick and after it had been opened we were fearful that fistulization might ensue, but it did not occur.

The size of the cyst was quite variable in my cases. Case I showed by radioscopy that it was the size of a large orange, but at operation it was found to be the size of a fetal head. In Case II the diagnosis of cyst was not made at the first radioscopic examination, but at the second, a shadow extending over a large portion of the lung was seen and mistaken for a large pleural collection. In Case III the diameter of the cyst was estimated to be about fifteen centimetres. Case IV was thought to be about the size of a small orange, but at operation it was much larger, while in Case V it was estimated that the hydatid might measure the size of a one dollar silver piece in diameter.

Case I was the only case in which numerous daughter cysts were found in the midst of a collection of pus. The other three operated cases had none.

It is especially necessary to bring out prominently two pathologic data which dominate clinically and reveal the genesis of the complications and throw light on the treatment of hydatid cysts of the lung. I refer to opening of the bronchial tubes into the pericystic cavity and the existence of a conjunctivo-vascular shell. In four cases pneumotomy clearly revealed the communication between the pericystic cavity and the bronchial tubes. In the fifth case a vomit leads me to conclude that an opening into the bronchi occurred, while in Cases I and III the hydatid expectoration left no doubt as to this occurrence.

All my patients presented hemoptysis in variable frequency and amount during the evolution of the cyst. Here are some details in respect to the anatomical conditions of broncho-cystic fistulization and hemoptysis.

According to Behr the opening of the bronchi into the cystic cavity is always lateral and as if bevelled; on the contrary, in a tuberculous cavity the bronchial opening being the result of a neoplastic process takes place directly into the lumen of the tube.

Dévé's experiments on sheep show that the perforation is often bell-shaped and rounded and that the bronchus opens into the cystic cavity by a clean-cut orifice.

Lepicard likens hydatid cysts to encysted empyema. Those of the greater cavity push the lung toward the mediastinum and only tardily rupture into the bronchi, while in the case of interstitial pleurisy in proximity to the interlobar bronchial quadrant, rupture is constant and takes place early. Hence the distinction of hydatid cysts into two categories, *viz.*, cortical or parapleural and central or parabronchial.

The cyst easily pushes aside the soft, elastic pulmonary parenchyma, but it exerts compression on the bronchi having a relatively large calibre. Dévé refers to this, in respect to the liver, as follows: "While the compressed blood-vessels have a general tendency to symphysis and occlusion, the bile ducts, thanks to their size and the mucosa lining them, usually remain patent in the midst of the fibrous tissue up to the point of contact with the parasite."

In the lung the factors of opening of the bronchi by compression are: (1) *The relatively large calibre of the bronchial canalization;*

(2) *the resistance of the fibro-cartilaginous framework of the bronchi*, which keeps these tubes widely gapping.

The virtual perivesicular space, which will become the cystic cavity after evacuation of the parasite, may become, on account of the bacteria coming from the bronchi, the site of a septic process.

The intact—hence aseptic—vesicle is surrounded by a tissue which may present an inflammatory process. At the point of contact with the bronchial openings, the eccentric pressure pushes back the membranes like a small hernia. Dévé removed a pulmonary hydatid cyst in which there were two vesicular hernias covered by a plug of bronchial muco-pus.

Hence may be explained vomit by traumatic shock, cough or compression. That the cyst partially empties, or sudden relief is obtained by incision or puncture, the hernia developing into the bronchus distends and ruptures, and the occurrence of operative vomit is explained. This is a clinical problem that can find its answer in a point mentioned by Dévé. In Case III, the patient's antecedents revealed that the bloody expectoration was occasionally accompanied by the presence of transparent pellicles, similar to "grape-skins."

Now, at operation, no daughter vesicles were found and the fluid was limpid. Where did these bits of membrane come from?

Lepicard states that on account of neighboring bronchial infection "after a time the external lamellæ soften and locally the wall loses its resistance on account of the endocystic process." The hydatid expectoration may perhaps sometimes be merely an exfoliation of the softened cuticular surface and on the other hand, Escudero states that all about the cyst, in the atelectasic or sclerous area according to the case, a rich network of vessels exists, some of which come in contact with the hydatid membrane.

Traction on and ulceration of these vessels are the causes of the slight hemoptyses noted in the vast majority of cases. When the adventitia becomes infected by bacteria from the bronchi it becomes infiltrated with leucocytes and offers a granulation tissue very rich in neo-capillaries.

The severe hemoptyses are due to septic ulceration of a vessel of some size—bronchial or pulmonary arteries or veins.

## THE SPONTANEOUS EVOLUTION OF PULMONARY CYSTS

A living cyst does not encounter any very considerable resistance to its eccentric expansion from the pulmonary parenchyma. Hence it may assume very large dimensions. Daibez has reported a case in which the cyst had reduced the lung to a shell one centimetre thick and Reid a case of necrosis of the ribs due to compression from an hydatid cyst of the lung. In most cases, however, the cyst becomes arrested in its eccentric development by one of the following eventualities, the first of which is

*Death of the Hydatid.*—In some infrequent cases the cyst withers, the fluid contents become thick and cloudy and the membrane becomes infiltrated with lime salts. Such a retrogression is more especially met with in hydatids of the liver, but typical examples are reported in the case of the lungs. In one of Guimbellot's cases he was able to follow this process in the lung by radioscopy; the distinct shadow was replaced by a smaller one with diffuse contours and dark offshoots giving the cyst a stellate aspect.

At the autopsy of a cattle-man, *æt.* 68 years, dying from influenzal broncho-pneumonia, Dévé discovered a puckered cicatrix on the external aspect of the middle lobe of the right lung, in the centre of which was a whitish indurated node the size of a filbert. In the midst of calcareous concretions he found small membranes which microscopically offered the characteristic refringent striation of hydatid membrane. No hooklets or scolex were discovered.

*Suppuration of the Cyst.*—Inflammation of the pericystic cavity from an infection derived from the bronchi may be transmitted to the cyst itself. The membranes exfoliate and allow the bacteria to enter the fluid contents of the cyst which are favorable culture media. The hydatid will then represent an encysted abscess.

Suppuration of pulmonary hydatids is common, having taken place in three out of my five cases. In one it caused repeated vomit, in the other two it was found at operation.

*Rupture of the Cyst.*—This usually takes place into the bronchi. When the cyst is central vomit occurs early in the evolution of the process, while in cortical cysts, on the contrary, it occurs late and then only when the hydatid is very large. Vomit occurs whether or not there is suppuration. In Case I the expectoration was purulent, the

pus containing grayish, fetid débris of the daughter cysts. In Case II the vomit took place during operation, while in Case V it was small in amount and perfectly limpid.

Rupture into the pleura is less common. Should it occur into both the pleura and the bronchi at the same time pneumothorax ensues. Rupture into the pericardium, spinal canal or intestine is exceptional but possible.

*Grafts.*—These will be a consequence of rupture and require both vitality and fertility of the cyst. Hence they do not take place when the cyst is suppurating. According to Dévé this explains the rarity of grafts in pulmonary hydatids. For grafts to ensue there must be a fissure in—exploratory puncture—or rupture of the cyst.

The forms of reproduction of hydatids within the human organism are the daughter vesicles, the proligerous capsules contain the scolex. Lebedeff von Alexinsky and Dévé have shown the possibility of secondary infestation by the scolex. Other observers have since definitely established the part played by this form of reproduction in hydatid grafts by experiments with injections of hydatid sand. From the lung, secondary infestation may take place by different routes.

*The Bronchial Route.*—Dévé has proved this experimentally and a fine illustration has been reported by Abadie of Oran. The cyst which was located in the base of the left lung ruptured and gave rise to two cysts in the left apex and to several other cysts in various viscera by way of the blood.

As in the case of the primary cyst *the circulatory route* is probably the most important of any and explains the hepatic, renal and splenic localizations in Abadie's third case.

No case has ever been reported in which the lymphatics served as a route for the dissemination of hydatids.

#### THE CLINICAL ASPECTS OF PULMONARY HYDATID CYSTS

If in a few words the cardinal semeiological characters presented by my five patients are to be described it is evident that all presented hemoptysis and that four had the characteristic expectoration. Two functional symptoms, *viz.*, hemoptysis and vomit usually denounce the presence of pulmonary hydatid cysts.

In respect to hemoptysis, Guimbellot remarks that "it is an almost

constant symptom, more or less copious, often repeated and occurring without any appreciable cause." Now in fact my patients either presented hemoptoic sputum or more abundant hemorrhage than Dieulafoy compared to "a bloody ejaculation," but in several of Lemaire's patients this symptom was absent. However, pulmonary hydatid cysts that have never given rise to hemoptyses may be considered as rare.

In Case I they occurred rather late in the process, at a time when puncture had already clinched the diagnosis, but when they did take place they were quite severe. When the patient did not have any notable hemoptysis she very frequently expectorated blood-tinged sputum. In Case II the hemoptyses took place early in the evolution of the cyst and were sufficiently severe to cause an amenorrhœa of seven months duration. During the entire clinical history they were repeated with varying intensity. At the time pneumotomy was done a purulent blood vomit ensued.

Hemoptysis was also the initial symptom of the onset in Case III. The contrast between the frequency of bloody expectoration and the absence of evidences of pulmonary tuberculosis were the reasons by which a correct diagnosis was reached. Thirteen days after the interference a violent hemorrhage occurred in the wound accompanied by an abundant loss of blood by the mouth.

The patient described as Case IV had been previously operated on for an hydatid cyst of the liver and the appearance of hemoptyses immediately led to the diagnosis of a pulmonary focus of the parasite. Finally in Case V hemoptysis was the initial symptom. Three times the loss of blood was severe but subsided quite easily after a more or less lengthy period of hemoptoic expectoration. The third hemoptysis was accompanied by a vomit of limpid hydatid fluid.

The essential point in these hemoptyses is that they appear suddenly without any apparent cause. Rest has not the same effect on them as in tuberculosis, while the exhibition of emetine usually controls the situation fairly quickly.

Hence this symptom is of great value. The frequency of hemoptyses coinciding with an absence of signs of tuberculosis should immediately lead the clinician to suspect the possible existence of an hydatid cyst of the lung.

The repetition and severity of the hemoptyses influence the patient's general health. Emaciation and cachexia, although very pronounced in Case I, in which the cyst had opened into the bronchi and abundantly suppurated, were less marked in the others. In Case IV the patient improved after removal of the hepatic hydatid, but the health again declined as soon as the pulmonary cyst manifested itself. On the other hand, in Case V there was hardly any anæmia in spite of hemoptyses and a vomit.

Guimbellot believes that the health remains good and that both the appetite and weight are preserved as long as the cyst remains intact without suppuration. This opinion, already to be found in text-books, may present exceptions. In Case III there was marked emaciation although the cyst had not ruptured and hence did not suppurate. It is hard to suppose that the general health can remain indifferent when hemoptysis is both severe and frequent.

*The Vomit.*—As to this occurrence during the clinical evolution of pulmonary hydatids in two of my patients it was purulent containing daughter vesicles and in one it was limpid. Muco-purulent expectoration from bronchial irritation is almost always present and Case V offers a very fine example. Developing after the first hemoptysis the expectoration of greenish thick putrid sputum, coinciding with signs of pericystic congestion will probably sooner or later cause cachexia when suppuration of the cyst ensues, although the child was in good physical condition when he left the hospital.

As to the expectoration of transparent pellicles as occurred in Case III where the pulmonary cyst, although close to the bronchi, had not ruptured and contained a limpid fluid without daughter vesicles, I have given the interpretation of this symptom.

The other symptoms—pain with radiation to the shoulder, dry paroxysmal coughing and effort dyspnoea—are common to other affections; they are also variable and they were noted in various degrees of intensity in all my cases.

Three of my patients presented urticaria and this symptom, although rare, is, when it exists, of utmost diagnostic importance. It usually occurs late in the evolution of the cyst and exploratory puncture frequently provokes its development; but in Case V no puncture had been made.

Escudero has pointed to the fact that in cysts near the pleura there may be pyrexia even when there is no infection. This febrile reaction is due to an intoxication from exosmosis of the fluid contents of the hydatid cyst. This took place in three of my patients, two in which there was suppuration and one where the cyst was unruptured. In Case V the slight pyrexia might have been due to a pericystic or bronchial infection.

The various symptoms that I have considered in reality do not offer—apart from a vomit containing membranes and hooklets—any absolute proof of the existence of an hydatid cyst of the lung.

Can a diagnosis be made by an objective examination? It is said that in voluminous cysts an arching of the thorax is present, percussion will give dullness, palpation reveals absence of vibrations and auscultation a marked decrease of the vesicular murmur. The thoracic arching is dome shaped, the dullness circular surrounded without transition by a sonorous area, auscultation reveals an area of alveolar impermeability, likewise round with its convexity toward the periphery. Sometimes egophony or aphonous pectoriloquy may exist.

In practice these findings rarely impose a diagnosis and Tuffier and Martin have aptly said that “the best proof of the relative importance of these signs is that the most eminent clinicians have only made the diagnosis by means of exploratory puncture in the great majority of cases.”

One reason that the diagnosis is so difficult is that the process being quite rare—all things considered—is not thought of, so that the physician interprets the physical signs presented as belonging to some one of the more common affections.

For that matter, although in all cases one finds dullness, absence of vibrations and decrease of the vesicular murmur, different modalities exist depending on the situation of the cyst and concomitant intrathoracic lesions that may exist. According to the size of the cyst, the thickness of the pulmonary parenchyma surrounding it, its anterior or posterior situation, the dullness will vary in intensity, the absence of the vibrations will be more or less complete and the diminution of the vesicular murmur will be more attenuated. When

a congestive process arises in the pericystic structures superadded signs ensue, such as râles, etc.

All that has been said applies to closed cysts. When rupture has occurred one may find tympany, cavernous or amphoric breathing or even all the symptoms of pyo-pneumo-thorax. Fine examples of this condition have been reported by Dévé, Olivier and Cerné.

And what is more, veritable complications may arise. For example, a pleuritic reaction from proximity, such as the experimental hydatid pleurisy with intense eosinophilia of the liquid produced in the monkey by Dévé, or Faisans' case in which a serofibrinous pleurisy was the means of calling attention to a latent pulmonary hydatid cyst on account of an eosinophilia having the cytologic formula of these cysts. Case V offers another example of this reactional serofibrinous pleurisy from proximity to the pulmonary lesion.

Finally, the coexistence of affections of various kinds, such as tuberculosis of the apex, pneumonia, broncho-pneumonia, aortic ectasis, etc., may also modify the clinical aspect. Hence this cluster of objective signs becomes singularly complicated and it is therefore easy to understand how a physician may be led to diagnose tuberculosis, pleurisy, broncho-pneumonia, etc., in a case which is in reality a hydatid cyst of the lung.

Let me rapidly refer to the objective symptoms present in my cases as well as those that were wanting. Only one of my patients presented a distinct arching of the thorax and this occurred late in the evolution of the process at the same time that other physical signs and pyrexia appeared which led to the supposition that there was a purulent pleurisy. The arching occupied the entire left hemithorax, especially behind and on the side. The hydatid thrill looked for when the real diagnosis had been made was never positive in any of my cases, but percussion gave dullness in all. In one case it was at the right base behind; in another there was dullness over the left base and slight dullness over the right apex. In the third case there was dullness over the middle of the thorax especially in front, not so marked behind. In the fourth case there was dullness over the left base, while in the fifth case there was slight dullness over the right base which varied on account of the pleural collection and the more or less congested state of the subjacent lung.

Search for vocal vibrations in the thorax gave variable data. In Case I at the beginning the vibrations were distinctly diminished in the area of dullness and afterwards were heard equally throughout the right lung. In Case II they were decreased in the dull area and exaggerated in the area of slight dullness in the right apex. In Case III they were decreased in front over the right base, in Case IV they could be heard throughout both lungs, while in Case V they were diminished, especially when the pleural collection developed.

The stethoscopic signs in Case I were congestion râles, pleural friction sounds and persistent vesicular murmur. In Case II the murmur was very diminished and inspiratory râles were heard throughout the left lung. Case III had a rough respiration at the apex, an intact impermeability at the base and respiratory obscurity at the middle. Case IV offered a whistling respiration in the right apex, and respiratory obscurity at the right base. Case V had a decrease of the vesicular murmur, friction sounds and inspiratory râles.

The perusal of the case histories will show the difficulties of clinical diagnosis better than a summary of the symptoms. For example, in Case I, the first physician to see the patient found signs of pleural fluid and made an exploratory puncture withdrawing some liquid, the character of which did not lead to a diagnosis, which was made at hospital. Radiography and radioscopy verified the clinical diagnosis, and the evolution of the cyst was watched. It then suppurated, giving rise to repeated vomit. It is to be noted that no signs of a cavity were detected at any time.

Case II is still more interesting, from the viewpoint of the clinical evolution. An aortic ectasis with insufficiency drew attention. The circulatory accidents explained the frequent hemoptysis and caused the angina pectoris and dyspnœa. Then signs of a fluid collection appeared, and it was regarded as a left hydrothorax from cardiac disease. Exploratory puncture withdrew nothing. It was only when pyrexia occurred and the signs of pleural fluid became more marked that a second puncture was made, this time giving exit to pus. Operation affirmed a diagnosis that even radioscopy—on account of the expansile shadow of the aorta and globular heart—had not suggested.

Case III is a typical example of hydatid cyst of the lung because

of its seat in the middle lobe. The physical signs showed beyond question that the right apex and base were intact. Hence the diagnosis was reduced to either an interlobar pleurisy or cyst. The hemoptyses, inexplicable by any other hypothesis, were in favor of cyst because of the translucent pellicles contained in the blood.

In Case IV the diagnosis was easy because of the previous existence of the hepatic cyst, while in Case V the early hemoptysis at first led to the suspicion of tuberculosis, but the disappearance of the signs of congestion at the left apex, then the symptoms developing at the left base followed by a vomit, led to the diagnosis of hydatid cyst.

Briefly, the protean character of pulmonary hydatid cysts and the rarity of this affection are the reasons why its clinical diagnosis is so difficult. An unruptured cyst will hardly ever be clinically recognized and usually exploratory puncture will be the only means for clinching the diagnosis. An open cyst has one pathognomonic sign, *viz.*, vomit or hydatid expectoration and even then one must know how to look for it.

#### THE VALUE OF THE OBJECTIVE METHODS OF DIAGNOSIS

There is one means by which an early diagnosis of hydatid cyst of the lung can be made, namely, puncture; the disadvantages of which are known to all. Exploratory puncture is more dangerous than puncture for evacuation. It can provoke a violent anaphylactic intoxication with rapid death, or a mild one with urticaria. It not infrequently gives rise to a vomit, the tissues traversed by the needle may be inoculated with the elements of the hydatid, and even when carried out with every aseptic precaution there is danger of infection from the bacteria of the bronchi or pericystic area, the fluid contents of the cyst being an excellent culture media. Therefore, puncture should never be done unless an immediate operation is to follow.

There remain the laboratory procedures, the first of which is *the examination of the expectoration*. This will be valuable only when the cyst has ruptured. It is clear that when hooklets are found in the sputum or in the vomit with striated membranes the diagnosis is made. The possible cause of error is the vomit from a cyst of the convex aspect of the liver which has opened into the bronchi.

*The humoral reactions* are numerous: Passive anaphylaxis (Chauffard and Boidin), the meiostagamine reaction (Ascoli) and the antitryptic index (Weinberg). But let it be said that with the exception of the latter which, when the index is high, indicates suppuration of the cyst, the others are no longer employed on account of their uncertainty.

The diagnostic procedures now current are:

The eosinophilia of the blood;  
Fleig-Lisbonne precipitio-diagnosis;  
Weinberg-Parvu fixation reaction.

The eosinophilia test of the blood is very simple. A drop of blood is evenly spread on a glass, fixed with alcohol-ether and stained with Giemsa or hematein-eosin. The percentage varies from 4 to 57 per cent. Boidin and Fiessinger found a local eosinophilia composed of acidophile mononuclears in the pericystic membrane. In most cases this local mononuclear eosinophilia becomes general, the mononuclears becoming polynuclears, a fact which explains the rapid disappearance of the eosinophilia after death or suppuration of the hydatid.

Although the eosinophilia is usually parallel with Weinberg's reaction, it may become dissociated. It is sometimes absent in hydatid cysts and it may be present in various other parasitic infestations. Lesieur, Rocher and Aigrot found it present in the pleural fluid in a case of gastro-hepatic cancer. Hence the conclusion must be accepted that eosinophilia cannot be absolutely relied upon for the diagnosis of echinococcosis.

Fleig and Lisbonne searched for the presence of precipitins in respect to the hydatid fluid in the serum of a patient with echinococcosis. Twelve drops of the blood serum to be tested are mixed in a tube with 1 c.c. of hydatid fluid and then sealed and sterilized in ampoules. The mixture heated at 40° to 50°C., should at the end of six to seven hours give a floccular precipitate which collects at the bottom of the tube. This reaction is specific and is fairly reliable. Out of eleven cases these observers found it positive eight times, once negative (suppurating cyst) and twice doubtful.

Walsh, Chapman and Storey found it positive six times and once doubtful in the blood serum and fluid of the cyst. Abadie, of Oran,

reached the following results: Of twelve tests, eight were positive in hydatid cysts of the liver, three times negative in other morbid processes (control cases) and once negative in a suppurating cyst. He believes that the yellowish color of the precipitate may be regarded as indicating suppuration of the cyst. All things considered, this method is quite reliable and technically easy.

The deviation of the complement—Bordet-Gengou method—applied to hydatid cysts was first tried out by Ghedini. Then Weinberg, of Paris, and Appathie and Lorentz, of Buenos Aires, advised the reaction of fixation. But this diagnostic procedure, like the search for eosinophilia, is not reliable. If these two tests are negative they are not conclusive and when positive they merely indicate that echinococcosis exists in some part of the body.

In *radiography* we have a sure method of diagnosis, but this should always be preceded by radioscopy. When a distinct shadow has been found, radiography is done. The patient, either standing or sitting, should keep the thorax erect, and the ampoule of the instrument placed first in front, then behind and afterwards laterally so that the lesion can be examined from every angle and the various details of the morbid process brought out.

In these circumstances radioscopy will detect pulmonary cysts even when small, hence permitting one to make a positive diagnosis. The cyst offers a nearly uniform opaque shadow standing out against the translucent pulmonary area. Its contours are rounded, with the convexity toward the periphery. The shadow will vary according to the vacuity or plenitude of the cyst. When the cyst has opened into the bronchi the part containing air will be clear, the dark line offered by the fluid always remaining horizontal no matter what lateral movements may be given to the thorax.

The exact site of the cyst can be determined by the relations of the shadow to the ribs, sternum, spine, clavicle and scapula, while its relations to the hepatic, diaphragmatic and cardiac shadows are easily seen. By changing the position of the ampoule one can determine whether the cyst is central, peripheral or nearer the anterior or posterior pulmonary surface.

But besides these diagnostic data, radioscopy will allow the clinician to make a differential diagnosis. The only intrathoracic affec-

tion that may cause confusion is aneurysm of the aorta. A free pleural collection will give a curve having its concavity uppermost that cannot be mistaken for an hydatid pulmonary cyst, while the displacements of the shadow will clinch the diagnosis. Escudero has called attention to the fact that in deep inspiration, and in certain positions the cyst, although usually immovable, may become displaced. An encysted pleurisy examined at different angles will invariably give a flattened shadow.

Pulmonary tuberculosis gives a diffuse shadow with ill-defined contours, usually festooned with irregular offshoots. The less intense opacity offers sudden variations.

Likewise in the case of a tuberculous cavity—for that matter usually multiple—there is not the dark regular ring of an hydatid cavity; the central luminous area is bordered by a rough outline. These lesions are almost always met with in the apices.

Cancer of the lung does give shadows similar to those of tuberculosis but still more irregular.

The genesis of these various shadows is easy to explain. An hydatid cyst pushes aside the pulmonary parenchyma by eccentric pressure without contracting any intimate union with it, while a tuberculous or cancerous process acts by neoplastic infiltration.

Lastly, there are the tracheobronchial lymph-nodes giving rise to polycyclical outlines on each side of the spine at the level of the hilum. These lymph-node opacities often coincide with tuberculosis or even hydatids of the lung.

Briefly, radioscopic examination makes more precise the data obtained by a study of the symptomatology, hence rendering the diagnosis almost a certainty.

Let me briefly refer to my cases in this respect. In one of them an aortic ectasis hid the pulmonary hydatid.

In Case I the radiological examination done on October 11, 1916, revealed a rounded shadow on the right above the diaphragm. This shadow had distinctly limited contours at the upper part while below they gradually extended to the hepatic shadow. On the right and left of the spine at the level of the hilum was a polycyclical shadow of tracheobronchial adenopathy.

Regardless of suppuration of the cyst and its rupture into the

bronchi by repeated small vomits, the second examination did not reveal collapse of the cyst. The diameter was that of a large orange and the shadow had retained the same appearance. After the operation radioscopy revealed the presence of the small tracheobronchial shadows. In the area of operation there was a slight diffuse shadow filling up the right costo-diaphragmatic sinus, the only trace of the cyst.

Speaking of the differential diagnosis between aneurysm of the aorta and hydatid cyst of the lung, Guimbellot states that "first of all that of the aortic arch must be considered, but its median site and movements of expansion and its continuity with the aorta, seen by radioscopy, will leave no doubt as to the true nature of the lesion."

Now the report of the radioscopic examination of Case II done on October 13, 1916, states that the left heart was enlarged and an aortic ectasis overlapping on the left. In fact the data obtained were as follows: At the upper part to the left of the spine was a shadow animated by expansile movements synchronous with the cardiac systole and encroaching upon the clear area of the pulmonary parenchyma. Below, the cardiac shadow itself appeared to be enlarged and encroached upon the left. This shadow presented beats likewise synchronous with the cardiac systole.

Given the existence of physical signs obtained by clinical examination of aortic insufficiency, the most logical interpretation of the globular enlargement of the cardiac shadow would be dilatation of the left heart, so that in this case the great difficulty, not to say impossibility, of reaching a conclusive diagnosis is readily conceived.

The second radioscopy done six months later—March 15, 1917—revealed a marked change; the pulmonary area on the right strikingly contrasted with a shadow involving the entire left lung. Now, as at this time the patient presented all the symptoms of an intrathoracic fluid collection, the interpretation of this shadow was not doubtful. Operation done three days later for a purulent pleurisy—pus had been withdrawn by exploratory puncture—revealed the true condition of affairs.

Radioscopy, done on June 12, showed the expansile shadow of the aortic ectasis extending one finger's breadth to the left of the sternum. To the left of the aneurysm one saw a denser shadow the size of an

average finger-nail which followed the movements of expansion and when the projection was varied seemed to be included in the wall of the aorta. The cardiac area was normal, the lungs were clear and there was no tracheobronchial adenopathy.

In this case radioscopy was insufficient for clearing up the diagnosis, but it must nevertheless be recognized that the circumstances which concealed the evolution of the cyst are quite extraordinary and in no way diminish the diagnostic value of radioscopy.

In Case III the shadow was typical of hydatid cyst of the lung (see Fig. 1). The left pulmonary area was perfectly clear. On the right a distinctly circular shadow was seen standing out in the midst of the clear pulmonary parenchyma. The apex and base were perfectly intact, the upper limits extended just above the fourth rib, the lower limits extended just below the eighth rib. By varying the patient's position the shadow was the most distinct when the patient's back faced the ampoule, hence we concluded that the cyst was near the anterior surface of the lung. Radioscopy done several months after operation revealed a very slight shadow, the only trace left of the former cyst.

Case IV was examined on March 28. On both the right and left were shadows of tracheobronchial adenopathy. On the right at two fingers' breadth above the diaphragm a rounded shadow was seen, about the size of a small orange. It was much more distinct when the patient faced the ampoule, hence the cyst was near the posterior surface of the lung. Although round in outline the shadow was very opaque in the centre and became less so toward the periphery.

Radioscopy done on July 2 showed that the operated lung was a little less translucid than its fellow. The shadow of tracheobronchial adenopathy was still present.

In Case V radioscopy permitted one to at once eliminate the diagnosis of tuberculosis, and, in spite of incomplete physical signs, led one to suspect a pleural collection. Successive examinations showed the persistence of the shadow of the cyst in spite of a vomit.

I would make the following remarks in respect to these cases. In spite of repeated vomits in Case I the cyst retained its initial appearance throughout. Guimbellot mentions the existence of obscure prolongations, giving a stellate shape to the shadow, due to sclerosis

resulting from the process of healing. In none of my four operated cases was such an arrangement found.

Although the diagnosis of aortic ectasis and pulmonary cyst is relatively easy by radioscopy when they exist separately, the same does not obtain when the two lesions coexist.

#### MEDICAL TREATMENT

It has been proposed to attack the cyst either by a supposed parasiticide general medication or by local treatment—intracystic injections. The attempts with serotherapy made by Dévé ended in complete failure, and this also applies to various antiparasitic remedies, such as calomel, sodium chloride, turpentine, iodine and various salts of Hg.

Kolbé, in 1914, taking into consideration the toxophorous action of arsenobenzol utilized successfully in spirilloses, trypanosomiasis and filariosis, thought that the drug might find a new application in the treatment of echinococcosis. Starting from the fact that retrogression of the cyst—when this does occur—takes place by necrosis and absorption, he suspected that the best medication would be the artificial production of these two phenomena, and he thought that intravenous injections of the arsenical product might be capable of causing necrosis and absorption of the cyst. He based his opinion on two cases reported by Roux, of Lausanne, who was able to extract by operation the necrosed membranes a week after an injection of 606. The only disadvantage of the arsenical product would be the too rapid necrosis of a very large cyst.

Wishing to verify Kolbé's assertion, Dévé carried out experiments in rabbits infested by echinococcus. He injected intravenously doses of arsenobenzol four times stronger than the maximum dose for an adult male. Upon three different occasions he injected six centigrams of 606 per kilogram of the animal's weight with the result that the echinococcal germs were in no way influenced. Hence Dévé concluded that the necrosing action of salvarsan and neo-salvarsan on hydatids is more than doubtful.

*Puncture and Injection of the Cyst.*—I have given the reasons why exploratory puncture should be discarded and treatment of pulmonary hydatid cysts by simple puncture has long since been given

up. *A priori*, puncture followed by injection of some parasiticide fluid might appear to be more logical as its aim is to kill the cyst directly. For this purpose a 1:4000 solution of Hg. bichloride, a 1 per cent. or 2 per cent. solution of formol or a solution of male fern have been used, usually in the following manner:

The cyst is punctured and a more or less large amount of its fluid contents withdrawn, this being replaced by an equivalent quantity of the formol solution. The liquid injected is withdrawn at the end of four or five minutes.

This procedure is useless. Perhaps the vitality of the parasite may be attacked but its spontaneous absorption does not ensue. That this is so has been amply demonstrated by numerous cases. For example, Gaillard injected 200 c.c. of a 1:4000 solution of Hg. bichloride upon two occasions and six months later the cyst had merely decreased a little in size.

Not only is this procedure ineffective, but above all it is *dangerous*. There is danger of a vomit with suffocation resulting from the sudden collapse of the cyst. Then there may be hemorrhage from sudden decompression from the vessels which frequently are ulcerated. There may be anaphylactic shock of varying gravity, even causing death. Hence puncture and injection should be discarded and in conclusion it can be said that *there is no medical treatment of hydatid cysts of the lung*.

#### SURGICAL TREATMENT

*Thoracotomy.*—The route of approach in my cases was always indicated by the data obtained by radioscopy. Only once (Case III), although radiography indicated the anterior approach as the shortest, the posterior approach was selected because a better declivous drainage could be obtained. During operation this choice was regretted because access to the cyst was insufficient, necessitating the resection of a second rib and making marsupialization difficult.

In none of my cases was a trap-door made and in three resection of one rib was sufficient. In respect to cysts of the lung I am of the same opinion as Professor Vincent, who, in speaking of hydatids of the pleura, says: "If the cyst is fairly small and limited to a spot of the pleural cavity, pleurotomy should be done directly over

the lesion; if the cyst occupies the greater part or the entire pleura, resection is to be carried out at the point of election in order to open the pleura in a declivous point, that is to say, at the level of the eighth rib, including the ninth as well, should this be necessary in order to obtain a better exposure or to drain the cavity at its lowest point."

It appears to me preferable to limit the extent of the operation and in reality *resection of a single rib is sufficient* in most cases even when the cyst is large, especially with the rib retractors at our disposal.

In my opinion an extensive thoracotomy should be reserved for very voluminous cysts in which it is probable that the cavity will not fill after operation or in cases of pachypleuritis. In Case I the ninth rib was first resected, but in order to obtain a larger field of operation, two vertical incisions were made at each end of the first incision in the eighth intercostal space.

In Case II the tenth left rib was excised and in order to enlarge the operative field the upper border of the incision was dissected up until the eighth intercostal space was exposed. In Case III the eighth right rib was resected and then in order to have more space a perpendicular incision four centimetres long was made at the anterior end of the first incision, which exposed the seventh rib, which was resected to the extent of about eight centimetres. In Case IV only eight centimetres of the eighth right rib were excised.

In all four cases the pleura was simply incised without costo-pneumopexia. Delagenière's technique was followed, namely, to make at first a small opening through which a finger was inserted to act as a valve regulating the entrance of air into the pleural cavity, but in no case was there a complete pneumothorax as the pleural adhesions prevented its occurrence.

When the cyst was exposed, pneumotomy was the most difficult in Case I. The pulmonary parenchyma was uniformly wine-red and was everywhere soft and elastic, and it was only by exploratory puncture that pus was located. Incision of the lung tissue gave rise to considerable hemorrhage, but this was controlled by ligature. The pus was readily let out and the hydatid membrane riddled with daughter cysts easily removed.

After the cyst was removed in all four cases the pericystic membrane could be examined and in all opening of the bronchi into the cavity was proven to exist. For this reason drainage was carried out and the operation completed by marsupialization.

Formolage of the cyst is dangerous and quite useless in the majority of cases because the cyst will have supplicated, therefore no graft is to be feared, and very frequently when suppuration has not occurred the lesion is an acephalocyst.

The prognosis of pneumotomy for hydatid cyst is good unless the operation has been delayed too long. Pasquier states that it is successful in 85.18 per cent. of the cases. Guimbellot has collected 223 cases of pneumotomy with 194 recoveries and 29 deaths, otherwise 87 per cent. successful results.

**A CASE OF TUMOR OF THE FOURTH VENTRICLE OF  
THE BRAIN, ACCOMPANIED BY CUTANEOUS  
PIGMENTATION LIKE THAT OF  
ADDISON'S DISEASE**

**BY F. PARKES WEBER, M.D., F.R.C.P.**

**WITH A REPORT ON THE TISSUES FROM THE CASE BY**

**HUBERT M. TURNBULL, M.D.**

**Director of the Pathological Institute of the London Hospital**

THE patient, L. R., aged 14 years, was admitted to hospital on December 31, 1920, suffering from nervous restlessness, excitement and irregular fever. The history was that for some weeks she had suffered from indigestion and want of appetite and tendency to "faintings." There had been vomiting during the last fortnight. A few days before admission she had fallen down on the stairs, and since then she had been very restless. Her father had been under treatment in the hospital during 1916 (when he was 52 years of age) with a positive Wassermann reaction and symptoms of tabes dorsalis.

The girl was very thin and had considerable cutaneous pigmentation, apparently of the type of Addison's disease, but the history was that she had always had a dark skin. Her blood-serum gave a negative Wassermann reaction. During the first days after admission her temperature varied between 98° and 101° F.; pulse, 92-120; respiration, 28-40 per minute. The urine (January 1st) was of specific gravity 1028, acid, and free from albumin and sugar. The brachial systolic blood-pressure (January 2nd) was 110 mm. Hg. Ophthalmoscopic examination showed nothing abnormal. There was more or less general hypo-æsthesia of the skin and pharynx. The knee-jerks could just be obtained. The plantar reflexes, when obtained at all, were of the flexor type. By abdominal palpation I found nothing abnormal, and by ordinary physical examination the thoracic organs appeared healthy. On a röntgen skiagram of the thorax (January 10, 1921) Dr. James Metcalfe reported: "String of small enlarged lymphatic glands at the pulmonary hilum on the right side, and to a lesser extent on the left side. The pulmonary apices appear quite clear, and there are no actual deposits seen in the lungs, but the translucency is somewhat deficient."

The restlessness gave place to mental apathy, drowsiness and feebleness. The irregular fever continued and on January 13th I noted that the pulse had lately been about 120; respiration, about 32-44 per minute. The patient had been having some potassium bromide, but this was discontinued on January 13th, and on the following day treatment by small subcutaneous injections of adrenalin chloride was commenced. A little port wine was likewise added to the diet. No improvement, however, followed. From January 17th to her death on January 29th the patient was more or less in a condition of stupor, and the temperature varied between 100.4° and 105° F. A second ophthalmoscopic examination (Dr. R. Gruber, January 17th) showed nothing abnormal. The brachial systolic blood-pressure at that time (January 17th) was found to be again 110 mm. Hg. On January 25th there were signs of pneumonia at the lower part of the left lung; pulse, 144; respiration, 56. She died about mid-day on January 29th. There was no acidosis; Gerhardt's and Legal's reactions were both negative in the urine of January 27th. The cutaneous pigmentation of the patient's trunk had apparently increased while she was in the hospital.

*Necropsy* (by Dr. Fankhauser).—In the fourth ventricle of the brain there was a tumor of about the size of a large acorn, attached to, and growing from, part of the wall (see further on, Dr. Turnbull's report). There was pneumonia of the lower lobe of both lungs. The suprarenal glands appeared somewhat small (but see further on, Dr. Turnbull's report). Otherwise no signs of disease were found. It should be especially noted that no evidence of tuberculosis in the lungs or elsewhere was discovered. The viscera were if anything rather small; the heart weighed only 6 ounces; the liver, 39 ounces; the spleen, 3 ounces; the kidneys together, 9 ounces.

The portion of the brain containing the tumor and both suprarenal glands were taken to Doctor Turnbull at the Pathological Institute of the London Hospital; who after very careful investigation kindly furnished me with the following report. An important point in the report, to which I will at once draw attention, is that Doctor Turnbull found no definite disease of the suprarenal glands. I suggest, therefore, that the remarkable pigmentation of the skin in this case may have been in some unknown way a result of the gliomatous disease of the brain (fourth ventricle).

## REPORT ON TISSUES FROM L.R., AGED 14 YEARS

By Hubert M. Turnbull, M.D.

Director of the Pathological Institute of the London Hospital

The following tissues were received in formaldehyde solution from Dr. Parkes Weber on January 31, 1921: (1) Cerebellum, pons and medulla. (2) and (3) Right and left suprarenal capsules.

## MACROSCOPIC EXAMINATION

(1) *Medulla, etc.*—The fourth ventricle has been opened by an incision through the vermis. This incision has passed through a rounded tumor which lies over the centre of the fourth ventricle.

The tumor measures 1.5 cm. from side to side, 2 cm. from before back, and 2 cm. from above down. It is attached to the mesial surface of the right superior cerebellar peduncle and to the floor of the fourth ventricle. The attachment to the right superior cerebellar peduncle is continued to the apex of the dorsal recess of the fourth ventricle, and thence onto the adjacent left superior peduncle for 0.3 cm. The attachment to the floor involves the whole of the right lateral recess and, diminishing in size rapidly toward its termination, passes to the left to terminate a short distance beyond the median groove, opposite and for 0.2 cm. behind the level of the left anterior fovea. The free, outer, convex surface is in close contact with the right posterior cerebellar peduncle, fills the left cerebellar recess and posteriorly lies against the nodulus.

The tumor consequently overhangs the fourth ventricle and fills the cavity with the exception of a small space over the anterior 1 cm. of the floor and a still smaller space over the posterior 0.5 cm. of the floor. The left half of the floor is concave and slightly flattened by the tumor.

A sagittal cut has been made into the tumor, exposing surfaces which are smooth, firm, elastic, homogenous and of the same color as the gray matter. The surfaces show one hemorrhagic area, 0.3 cm. in diameter.

A coronal section through the tumor, cerebellum and the medulla near its anterior extremity, shows that the tumor has not involved appreciably the central white matter of the right lobe of the cerebellum nor the gray matter of the floor of the ventricle. This cut

surface of the tumor is whiter, and shows two small cysts (the largest 0.3 in diameter) with smooth walls and watery content.

(2) and (3) *Suprarenal Capsules*.—The two capsules, fixed in formaldehyde, weigh together 10 grams after removal of the attached adipose tissue. The right measures  $4 \times 3 \times 0.5$  cm.; the left measures  $5 \times 2.3 \times 1$  cm. The cut surfaces show a cortex (0.15 cm. deep) of pale yellow, interrupted by a few areas of pale gray. In the central portion of the capsules the cortex encloses medulla of pale slaty gray color. Between the yellowish cortex and the pale gray medulla there is a line of darker gray color, which varies in breadth and occasionally shows a tinge of warm brown.

#### MICROSCOPIC EXAMINATION

(1) A segment of the tumor, the subjacent medulla and the adjacent part of the right lobe of the cerebellum were embedded in paraffin and sections were stained in Ehrlich's hematoxylin with eosin, Weigert's iron hematoxylin with and without van Gieson's mixture, and Weigert's fuchselin with neutral red.

(2) and (3) A segment from the centre of each suprarenal capsule was embedded in paraffin, and the sections were stained by the above methods, and with dilute carbol-fuchsin, neutral red, and Gram's stain with neutral red. Other segments were cut upon the freezing microtome, and sections were stained with Sudan III and hematoxylin.

(1) *Tumor and Medulla*.—The section of the medulla includes the principal vestibular nuclei, Deiters' nuclei, the right nucleus of Bechterew, the cochlear nuclei, the formatio reticularis, the olivary and accessory olivary bodies and the pyramids. The restiform bodies are elongated in the dorsal direction. Mesial to the dorsal extremity of the right restiform body is the ventral extremity of the right superior cerebellar peduncle. This peduncle forms the greater part of the right lateral wall of the ventricle; it is continuous with a portion of the right superior medullary velum. The tumor and a portion of the cerebellum are present on the right side. The tumor is truncated toward the left by the longitudinal incision which has been made through it in opening the fourth ventricle at the necropsy.

The tumor lies upon the floor of the fourth ventricle from the right lateral recess to the outer limit of the left dorsal longitudinal bundle. It is in direct continuity with the subjacent gray matter, except at a few spots at which it is separated by small clefts lined with ependyma. All these clefts, save one, lie close to the median sulcus. The tumor is in direct continuity with the right lateral wall of the ventricle to within a short distance from the dorsal extremity of the right superior peduncle. It is separated by a narrow cleft from the dorsal extremity of the right superior peduncle and the superior medullary velum. The free surface of these structures is lined by an almost complete layer of ependymal cells; the free surface of the tumor shows only a few groups of ependymal cells.

The tumor is composed of glial tissue. It contains perfectly formed capillaries, veins and arterioles with adventitial clefts; the tissue of the adventitia is frequently swollen and hyaline. In its central part and in its ventral part, where it is in contact with the floor of the ventricle, comprising in all about one-half of the whole cut surface, the tissue differs from normal glia only in that glial nuclei are slightly more numerous, in that the majority of the nuclei lie within recognizable cell-bodies, which are of either polygonal, stellate, pyriform or fusiform shape, in that the nuclei occasionally show karyorrhexis and karyolysis, and in that a few hyaline bodies are present. In sections stained with iron hematoxylin and van Gieson's mixture these bodies are yellow, or, occasionally, dark brown merging into yellow at the periphery; in Ehrlich's hematoxylin and eosin the corresponding colors are pink and purple.

The remainder of the tumor has a much coarser appearance when examined under a low power. This coarseness is due to the great number of similar hyaline bodies, and the great size of many of these bodies. The bodies lie in a net of glial fibrils furnished with nucleated glial cells, but the greater the number of bodies the fewer are the glial fibrils and cells. The bodies vary very greatly in size and shape. Many are cylindrical, and measure from 1 to  $7.6\mu$  in diameter and as much as  $70\mu$  in length. In places these cylinders are arranged in interlacing bundles. The cylinders are sometimes branched; those of large diameter are frequently moniliform or of elongated pear shape. Other bodies appear to have axes which differ

little in length. Such bodies are spherical, polygonal or pyriform; the spheres may measure as much as  $22\mu$  in diameter. There are a few bodies which consist of a collection of spheres and in a few cases these spheres are enclosed within a ring of similar substances. A cyst occurs in an area in which these various bodies are numerous and large. None of the bodies are nucleated.

In regard to the nature of these bodies, transitions can be traced between glial fibrils and the cylindrical examples of small diameter. The shape of the spherical and pyriform examples suggests an origin in glial cells, but the nucleated glial cells seldom measure more than  $15\mu$  in longest diameter. Other bodies suggest capillaries which have undergone a coagulative necrosis. Whatever the exact origin of these bodies may be, whether from alteration of formed structures or from precipitation of chemical substances in solution, there can be no doubt that they are products of degeneration.

With the exception of the few small clefts lined with ependymal cells, which have been mentioned above, there is no sharp differentiation between the tumor and the floor and the right lateral wall of the ventricle. The tissues merge into one another. Toward the left the gray matter of the floor of the ventricle is clearly differentiated by the greater density of its glial reticulum. Further to the right the presence of medullated fibres gives an indication of the line of junction with the floor and with the lateral wall of the ventricle. But occasional medullated fibres appear to be incorporated within the periphery of the tumor, at any rate some medullated fibres are in close association with the degenerative bodies which are so characteristic of the tumor.

The bodies of the neurons in the nuclei of the floor of the ventricle contain little or no neurochromatin. This, however, is true of the cells throughout the section, including the Purkinje cells. The cell nuclei are central. Judged by shape and the occasional presence of neurochromatin, the cells in the right vestibular nucleus and Deiters' nucleus, beneath the tumor, are as healthy as the cells elsewhere.

(2) and (3) *Right and Left Suprarenal Capsules.*—Degeneration of the tissue after death and before fixation is indicated by slight dissociation of the parenchymatous cells, swelling of the interstitial tissue and lysis of red corpuscles. In the capsule, in the interstitial

trabeculæ, in the capillaries and, to a less extent, within the cellular columns are great numbers of Gram-negative filaments, short stout bacilli and shorter diplo-bacilli. These all appear to be forms of the same organism. They contain unstained vacuoles, and are about  $0.75\mu$  broad. The individual bacilli of the diplo-bacilli measure about  $1\mu$  in length, the shorter bacilli  $2\mu$  to  $3\mu$  and the longer filaments  $10\mu$  or more. There are also many Gram-negative cocci and a few Gram-positive cocci and diplococci. The bacteria are more abundant in the outer portions of the sections than in the central. The infiltration with bacteria is not associated with any inflammatory reaction.

The cells of the medulla are large. Their cytoplasm is finely granular. Their nuclei, with very few exceptions, show no sign of degeneration.

Granules of yellow pigment are visible in the cells of the reticulate zone of the cortex in paraffin sections which have been very lightly stained with hematoxylin. In paraffin sections the cells in portions of the fascicular zone show a conspicuously vacuolated, "spongy" or "foam-like," cytoplasm. This vacuolation affects the central segment of the fascicular column, or the whole column with the exception of a few cells close to the glomerular zone; sometimes the reticular zone is implicated. Further, the vacuolation affects a variable number of adjacent columns, so that in stained sections under a low power of the microscope the areas occupied by vacuolated cells appear in the cortex as isolated pale patches which are either small and somewhat rounded, or extensive and oblong. In the remainder of the cortex the cytoplasm of the cells is granular, and only occasionally contains a few small vacuoles. In frozen sections examined with the polariscope or stained with Sudan, fatty granules occupy, and are confined to the vacuoles seen in the cells of the paraffin sections, and the fatty granules are birefringent. The lipin in the cortex is, therefore, confined to small rounded and larger oblong areas which lie in the fasciculate zone and occasionally implicate the reticular zone.

The nuclei of the cells in the cortex are not so well preserved as those of the cells in the medulla. Near the capsule they are frequently pale and without distinct threads of chromatin. This nuclear change is correlated with the presence of bacteria and doubtless expresses an agonal or post-mortem alteration.

## SUMMARY AND INTERPRETATION OF ANATOMICAL OBSERVATIONS

*The tumor* in the roof of the fourth ventricle consists of glial tissue in which the relative number of cells to fibres is only in slight excess of the normal. The structure is so "typical" that the term "glioma" is indicated in preference to "glio-sarcoma." The tumor exhibits evidence of considerable degeneration; this is in part cystic. As was seen with the naked eye, it is in direct continuity with the floor of the fourth ventricle, from the right lateral recess to the outer border of the left dorsal longitudinal bundle, and with the right lateral wall of the ventricle. The sections do not afford evidence that the tumor has caused degeneration in the adjacent nuclei of the medulla.

*The suprarenal capsules* weighed together, after fixation in formaldehyde, 10 grams. They have been invaded by various organisms in the death-agony or after death, and exhibit changes characteristic of putrefaction. Pigmentation of the reticular zone of the cortex is present, but is not easily detected in sections. Fatty substances are confined to focal areas, or patches which lie within the fascicular zone of the cortex and occasionally implicate the reticular zone. The greater part, if not all, of the fatty substances is birefringent.

In order to determine whether pathological changes are present, and to decide the significance of such changes, the suprarenal capsules from four subjects of approximately the same age were examined as controls. Two controls were taken to demonstrate the structure of the capsules in healthy individuals; two were taken to demonstrate the changes likely to be caused by a general infection. These controls were:

- (1) Male, aged 14 years. Accident. (2) Male, aged 14 years. Accident. (3) Female, aged 15 years. Streptococcal peritonitis. (4) Male, aged 15 years. Staphylococcal pyæmia; osteomyelitis.

In regard to the weight of the two suprarenal capsules it was first ascertained that on the balance employed there was no appreciable difference in weights estimated before and after fixation of the capsules in formaldehyde. The weights in the above controls were as follows: (1) Body: 88 lbs. 7 oz.; capsules: 10 grams. (2) Body: 87 lbs. 1 oz.; capsules: 6.5 grams. (3) Body: 97 lbs.

14 oz.; capsules: 13 grams. (4) Body: 81 lbs. 10 oz.; capsules: 18 grams. According to these controls the weight of 10 grams is not obviously abnormal.

In histology the only differences are found in the amounts of lipinous content. In the two healthy subjects (Controls 1 and 2), the cells of the fasciculate zone of the cortex, and often the cells of the glomerular and reticulate zones, show a spongy cytoplasm in paraffin sections, and in frozen sections are loaded with birefringent, sudanophil bodies. In Control 3, in which death was due to streptococcal peritonitis, only occasional and small groups of cells in the fascicular zone, or, rarely, in the glomerular and reticular zones, have this spongy cytoplasm and contain birefringent sudanophil bodies; there are also a few large globules of isotropic fat. The amount of lipin in the capsules from this control is far less than in the capsules under discussion. In Control 4, in which death was due to pyæmia, the amount of lipin is still less; sudanophil bodies are confined to extremely few groups of cells in the fasciculate and glomerular zones; the groups are seldom composed of more than a dozen cells; isotropic globules are present as well as birefringent bodies.

The only abnormality discovered, therefore, in the suprarenal capsules when compared with controls from healthy subjects of the same age, is a deficiency in lipin. Such a deficiency is known to occur in general infections; a very much greater deficiency is present in a control from a case of streptococcal peritonitis, and a still greater in a control from a case of staphylococcal pyæmia. It is justifiable to conclude that the deficiency in lipin is the consequence of the terminal, fatal pneumonia, and cannot be held responsible for any clinical manifestations which preceded this terminal infection.

## THE DIAGNOSIS OF BRONCHIAL GLAND TUBERCULOSIS

BY JOHN B. HAWES, 2ND

Boston, Massachusetts

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I DO not know of any more difficult diagnosis than that of bronchial gland tuberculosis. In this statement I emphasize the word "tuberculosis," however, as the diagnosis of enlarged bronchial glands does not in itself show any inherent difficulties either with or without an X-ray examination. Add the word "tuberculosis" and the problem becomes a complex one and the truth difficult to find.

Within the past month I have seen three children from seven to twelve years of age in each of whom a diagnosis of *tuberculous* bronchial glands had been made or at least very strongly suggested. At all events, whether or not the physician intended to make the diagnosis a definite one the fact remains that in each instance the parents understood that their child had tuberculosis and naturally enough were greatly alarmed at the situation. A brief summary of these cases is as follows:

CASE I.—G. P. H., *æt.* 12. This boy had a grippy infection at age of five and ran a temperature for twelve weeks. January, 1921, he was in bed for two weeks with laryngitis and an X-ray at that time showed enlarged bronchial glands. Since then he has not been quite so well and has run a slight evening fever of 99 to 99.2. Physical examination was negative except for a very ugly looking right tonsil, which on being removed was found to contain nearly a teaspoon of pus.

CASE II.—M. K. S., *æt.* 7. This boy has never been very well since a bad attack of influenza in 1918. Tonsils and adenoids were removed in 1919 on account of repeated sore throats, but the boy still coughs, raises sputum and runs a temperature occasionally as high as 101 or 102. His mother would consider him well now except for fever. The X-ray taken in August, 1921, during an acute exacerbation of his symptoms showed enlarged glands and according to the röntgenologist "suggests the positive diagnosis of tuberculosis of the left lung." The Von Pirquet, which I did myself, was negative and to me at least the X-rays did not suggest tuberculosis of any kind.

This, in my opinion, is a low-grade pulmonary infection following influenza. It may be tuberculosis, but I doubt it. There is probably a mild bronchiectasis causing the cough and sputum.

CASE III.—C. N., *æ*t. 8. This boy began to run a fever up to 100° shortly after a tonsil and adenoid operation at the age of five. In addition to this, he began to show signs of undue fatigue and was taken out of school and has not been to school since. An X-ray taken last year showed enlarged bronchial glands. Another X-ray taken this year shows the glands to be still more enlarged. His physician in the country believed the glands to be tuberculous and told the mother so. One tuberculin test was negative and a later one positive. The boy complains of no symptoms, but, as a result of the invalid life he leads, is very fat and logy. Physical examination shows nothing abnormal.

In each of these children although the diagnosis of tuberculosis was a possibility it was so far from being a probability that I hardly considered the use of that particularly ugly sounding word to be justified. These children had been under observation a long period of time and had each been repeatedly examined including an X-ray. I think it was particularly due to the loose use of the term, "tuberculosis," by the röntgenologist which led the family physician to assume that the bronchial glands, undoubtedly enlarged, were so enlarged because of a tuberculous infection. These patients presented practically the same group of signs and symptoms, as follows:

1. Slight but fairly continuous evening fever, 99-100, with an elevated pulse with occasional acute exacerbations with sudden rises of both temperature and pulse.

2. Debility, lack of strength and endurance to a greater or less degree, particularly "ease of tire" along with a somewhat irritable nervous system.

3. A tendency to catch colds and to have such colds hang on.

4. Enlarged bronchial glands as shown by the X-ray.

In only one instance was there a positive skin tuberculin test. There was no loss of weight, in fact, quite the reverse was the case as on the whole each of these three children was rather fat and overweight. The question at once arises which I shall discuss here as to whether or not under these circumstances the use of the word, "tuberculosis," in describing to the parents the condition of their children was justified. Personally, I do not think that it was. I feel that we and particularly the X-ray men are using that term, "tuberculosis," in an extremely loose fashion so that to the parents it means disease when to us it means merely infection with the result that the parents infer that their child has something clinically wrong when we mean

something pathologically wrong. This does not help them nor the patient.

"Tuberculosis," "phthisis," and "consumption" are ugly sounding words. While a house officer at the Massachusetts General Hospital under the service of Dr. Frederic C. Shattuck, one of the greatest practitioners of the art of medicine that this country has produced, I was given a striking lesson on the effect that an ugly sounding word might have on the patient. As we were going through the ward I pointed to a certain bed saying, "This, Doctor Shattuck, is a case of pernicious anæmia." He stopped, led me away from the patient's bedside and remarked quietly in a way that I shall never forget, "*Primary* anæmia, Hawes, *primary* anæmia; remember that 'pernicious' has an ugly sound to the patient." The same thing applies to the word, tuberculosis. If we are absolutely sure of our ground, well and good, let us go ahead and speak plainly; if we have a patient who is hard to control and who must be "hit on the head," as it were, in order to make him do the proper thing, use the ugly word and emphasize it by all means but when we only suspect the truth I do not believe that it is wise or proper to do this if we can institute treatment and accomplish our aim in a gentler fashion.

Dr. George Porter, for many years Secretary of the Canadian Tuberculosis Association, as part of his duties was called upon to give lectures on the subject of tuberculosis throughout Canada. One year he was making a tour of small fishing villages along the coast of New Brunswick. He knew that he was speaking to people who were apprehensive of disease and to whom the words, "tuberculosis" or "consumption," would be a source of alarm and not of interest; he knew that if it were announced that he were to speak on such a subject that no one would attend his lecture. He described to me how he made a tour, going from village to village, speaking before crowded houses and how not once during his talks did he use the words, "tuberculosis" or "consumption." Yet I know only too well the lesson that he taught was a vivid and a valuable one and that he accomplished what he aimed to do.

Of the three children referred to above who were brought to me with this provisional diagnosis of bronchial gland tuberculosis I found that one had a tonsil which contained half a drachm of free pus and

which upon its removal has been followed by a subsidence of all symptoms. The second had a negative Von Pirquet test which I performed myself and upon further study has, I believe, a beginning bronchiectasis but no evidence of tuberculosis. He is now doing well. The third may possibly have bronchial gland tuberculosis but at all events the chief things that troubled him were that he was immensely over-weight and was not getting enough exercise or iron-bearing foods. He is steadily improving under a new regime.

Dr. Lawrason Brown speaking before the recent Tuberculosis Institute held by the Boston Tuberculosis Association in a scholarly address emphasized five points, at least one of which must be present, in order to make a diagnosis of pulmonary tuberculosis in the adult. These five points were as follows:

1. Tubercle bacilli in the sputum.
2. Persistent râles at the right apex above the second rib.
3. X-ray evidence of parenchymatous infiltration over the same area.
4. A history of hemoptysis amounting to at least one teaspoonful of clear blood.
5. A history of pleurisy with effusion.

These five points are not applicable to the diagnosis of tuberculous bronchial glands in children. In the Diagnostic Standards of the National Tuberculosis Association under the heading of "The Diagnosis of Thoracic Tuberculosis in Childhood" certain minimum standards are laid down. These minimum standards are of distinct value but their value is not so great since the recent epidemic of influenza as it was before that time. Influenzal infections of all kinds, both in children and in adults, have upset many of our standards on which we formerly based our diagnosis of tuberculosis. In view of the prevalence at the present time of small patches of broncho-pneumonia and other acute and subacute pulmonary infections not due to tuberculosis it is well to go over these diagnostic standards with care and see if we cannot lay down certain points corresponding to those of Doctor Brown. My own ideas of such diagnostic points would be as follows:

1. A positive skin tuberculin test unless the child has recently

recovered from measles or any of the other acute infections which might lead to a negative test.

2. Constitutional signs and symptoms, particularly loss of weight or failure to gain weight, along with "ease of tire" or undue fatigue, fever or rapid pulse.

3. The presence of enlarged bronchial glands as shown by X-ray or by clinical examination of the chest.

4. The absence of other evident sources of infection or toxemia such as,

- (a) Infected tonsils or adenoids,
- (b) Carious teeth,
- (c) Intestinal disturbances and particularly a chronic appendix,
- (d) Other possible sources of infection such as middle-ear, glands in the neck, broncho-pneumonia, etc.

5. A definite history of exposure from either human or bovine source of tuberculosis.

Of these five points I should say that a positive tuberculin test is the only one which must be present before the diagnosis of bronchial gland tuberculosis can be made. Constitutional signs and symptoms, however, as indicated above to a greater or less degree should likewise be present before a diagnosis of active disease is justified. A diagnosis is never justified on X-ray evidence alone nor as was apparently the case in the three patients above mentioned with the combination of X-ray evidence plus persistent fever and certain other constitutional symptoms.

I believe that bearing these points in mind we will come to more accurate conclusions in the diagnosis of this important but difficult problem. For instance, in one of the children that was brought to me an infected tonsil was the cause of the symptoms. This would have been discovered and ruled out had the five points that I have mentioned been borne in mind. In the second case although there was a history of constitutional signs and symptoms (without loss of weight, however) and X-ray evidence of enlarged glands, the skin tuberculin test was negative while the history pointed toward an acute pulmonary lesion as the beginning of the trouble. In the third case again, the tuberculin test was negative.

Summarizing these remarks I would emphasize the following:

1. The diagnosis of bronchial gland tuberculosis in children is a difficult one, involving serious responsibility on the part of him who makes it.

2. Bronchial glands may become enlarged and may be accompanied by symptoms from other causes than tuberculosis.

3. A positive skin tuberculin test must be present before such a diagnosis is justified.

4. Constitutional signs and symptoms, particularly loss of weight or malnutrition, should be present before a diagnosis of active tuberculous disease is justified.

5. The presence of enlarged bronchial glands may be shown by clinical or X-ray examination or both.

6. Bear in mind the other causes of enlarged bronchial glands such as teeth, tonsil, adenoid or other ear, nose or throat infections; pulmonary infections and acute or chronic infections of the gastrointestinal tract.

7. Institute proper treatment but be sure of your grounds before you use the word, "tuberculosis," in describing the glands.

## PREVENTION OF SPORADIC SIMPLE GOITRE

BY ISRAEL BRAM, M.D.

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IN contradistinction to *endemic* goitre, by which term is meant thyroid enlargement occurring constantly in certain districts, the term *sporadic* goitre indicates thyroid enlargement as a result, not of geographical conditions, but of causes known and unknown, occurring everywhere.

The management of simple or non-toxic<sup>1</sup> goitre has occupied the minds of many observers, eminently Marine and Kimball, Lenhart, Klinger, Hunziker, Kjalstad, and others, but their work was confined mainly to the endemic type of the affection. Although the endemic and the sporadic forms may appear alike from the viewpoint of the clinician, they should be sharply differentiated in our consideration of the patient from the viewpoint of therapeutics.

A person may reside in an endemic goitre district and yet never acquire an enlargement of the thyroid. This same person, sojourning elsewhere, may develop goitre through causes not peculiar to endemic districts. Contrariwise, a person with a well-developed sporadic goitre, on changing her residence to a goitrous district may find her goitre diminished in size or perhaps disappear. This desirable result is not due to change of residence, but to some natural physiological adjustment within the body itself, the individual being at the same time immune to geographical causes of thyroid enlargement.

In the distinction, then, between endemic and sporadic simple goitre, we must include a consideration of susceptibility to or immunity from, as the case may be, the etiological factors in goitrous districts on the one hand, and factors, not including geographical conditions, favoring the development of goitre, on the other. It is easy enough, in a non-goitrous district, to conclude that a given goitre is sporadic in nature. It is rather difficult and often impossible, to

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<sup>1</sup> The terms "simple" and "non-toxic" goitre are here used interchangeably, indicating a thyroid enlargement not accompanied by thyrotoxicemia.

make the distinction in patients residing in goitrous districts, as to whether the thyroid enlargement is induced by geographical conditions or otherwise.

#### IODINE IN SIMPLE GOITRE

The prophylaxis and treatment of endemic goitre is largely based upon the theory that there is a lack of iodine in the thyroid gland, and that, when the iodine in this organ reaches to or falls below .1 per cent., goitre develops. This lack of iodine in the thyroid is brought about by a deficiency or absence of iodine in one or more things essential to life's processes (air, water, or food), which deficiency or absence is peculiar to the district in question. Accordingly, it has been found that the administration of the proper quantity of iodine in some form to the growing child or adult residing in these districts is successful in the prevention and treatment of goitre in the majority of cases. In general, this is true of endemic, but not of sporadic simple goitre.

In the prophylaxis of goitre not evidently caused by geographical conditions, iodine administration, contrary to the prevalent opinion, is far from routinely successful. Of the comparatively small percentage of these patients who are helped by iodine therapy, it may be assumed that their habits of life, especially dietary habits, are such as to approximate in effects upon the body those conditions peculiar to geographical deficiencies. A few individuals may be so constructed as to require a greater quantity of iodine for the normal performance of the bodily functions than all the rest of humanity, and for that reason develop thyroid enlargement in the presence of an intake of what would ordinarily be considered a normal quantity of iodine. These individuals, though not living in endemic goitre districts, are scattered instances of pseudo-endemic goitre, the prevention of which should be based upon iodine therapy. But, to repeat, the percentage of such cases in non-goitrous districts, *i. e.*, among great numbers of sporadic simple goitres, is not as great as it is generally thought to be. To endeavor to prevent or to treat simple non-endemic goitre routinely through iodine administration yields an occasional success with a large percentage of failures, and in not an inconsiderable percentage of instances evident harm is wrought.

## COMPLEX ETIOLOGY OF SPORADIC SIMPLE GOITRE

The reason for failure and even harm resulting from the use of iodine in the prophylaxis or treatment of non-endemic goitre is not far to seek if we but remember the known etiology of this type of thyroid enlargement. Let us recall that sporadic goitres may be due to the following factors, none of which are dependent upon a deficiency of iodine intake:

1. *Heredity*, which seems to play an important rôle. In my observations, this seems true in at least 40 per cent. of patients. On glancing through my records at this writing, I find quite a number of instances in which there are three and four immediate members of a family presenting goitre.

2. *Acute infectious diseases*, especially acute articular rheumatism, influenza, and typhoid fever, may instigate goitre formation during, but more often following, the course of the affection. Such chronic infections as tuberculosis and syphilis likewise play an etiological rôle.

3. *Focal infections* from teeth, tonsils, nasal sinuses, and more remotely from the gastro-intestinal and genito-urinary tracts, are commonly responsible for thyroid enlargements.

4. *Puberty, adolescence, pregnancy, lactation, the menopause*, and even the ordinary menstrual function in established adult life, are productive of thyroid swelling in susceptible individuals. I say susceptible because, aside from hereditary tendencies, it is difficult to understand why one person does and another person does not, under the same circumstances, develop thyroid enlargement. There is a direct and striking relationship between the functions of the organs of reproduction and the thyroid apparatus. In this connection, we might state that

5. *Diseases of the female reproductive organs*, eminently ovarian and uterine conditions, are often etiologically responsible for goitre formation.

6. *Miscellaneous causes*, little known or unknown in nature, which, for want of a better heading, might be placed under that excuse for ignorance, the term "idiopathic," form another etiological group. Probably here may be included numerous instances of marked dietary

FIG. 1.



FIG. 2.



FIG. 3.

FIG. 4.

FIG. 1.—Endemic goitre of 21 years' duration.  
FIG. 2.—Endemic goitre of 40 years' duration.  
FIG. 3.—Endemic goitre of 40 years' duration.  
FIG. 4.—Beginning sporadic goitre in child of 4.

FIG. 5.



FIG. 6.

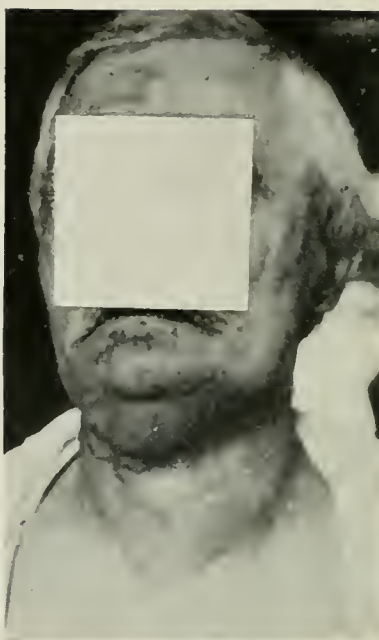


FIG. 7.



FIG. 8.

FIG. 5.—Sporadic goitre of 3 years' duration.  
FIG. 6.—Sporadic goitre of several weeks' duration in Civil War  
veteran of 74.  
FIG. 7.—Sporadic goitre in child of 7.  
FIG. 8.—Sporadic goitre in child of 12.

FIG. 9.



FIG. 10.



FIG. 11.



FIG. 12.

FIG. 9.—Sporadic goitre of adolescence; 2 years' duration.  
FIG. 10.—Sporadic goitre of adolescence; 3 years' duration.  
FIG. 11.—Sporadic goitre of about 14 years' duration (hereditary tendency).  
FIG. 12.—Brother of patient in Fig. 11 with goitre of 2 years' duration.

FIG. 13.



FIG. 14.



FIG. 15.



FIG. 16.

FIG. 13.—Sporadic goitre of 30 years' duration (hereditary tendency).

FIG. 14.—Daughter of patient in Fig. 13, with goitre of 4 years' duration.

FIG. 15.—Sporadic goitre of adolescence; 2 years' duration.

FIG. 16.—Same patient as Fig. 15, after 6 months of treatment; goitre nearly gone.

FIG. 17.



FIG. 18.



FIG. 19.

FIG. 20.

FIG. 17.—Sporadic goitre of adolescence; 2 years' duration.

FIG. 18.—Same patient as Fig. 17, after 6 months' treatment; neck normal.

FIG. 19.—Sporadic enlargement of right lobe of thyroid; 4 years' duration. (Note profile of neck.)

FIG. 20.—Same patient as Fig. 19, after 8 months' treatment. Thyroid is normal.

FIG. 21.

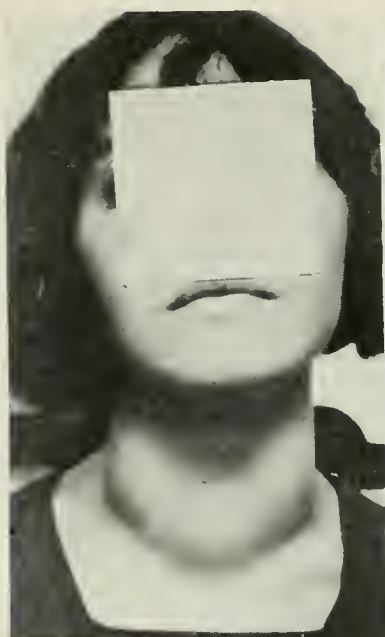


FIG. 22.



FIG. 23.



FIG. 24.

FIG. 21.—Sporadic goitre of adolescence; 2 years' duration. Circumference of neck  $15\frac{1}{2}$  inches.

FIG. 22.—Same patient as Fig. 21 while under treatment. Neck reduced to  $13\frac{1}{2}$  inches in circumference within three months and rapidly becoming normal.

FIG. 23.—Sporadic goitre of 4 years' duration.

FIG. 24.—Same patient as Fig. 23, after 4 months' treatment. Neck is normal.



FIG. 25.

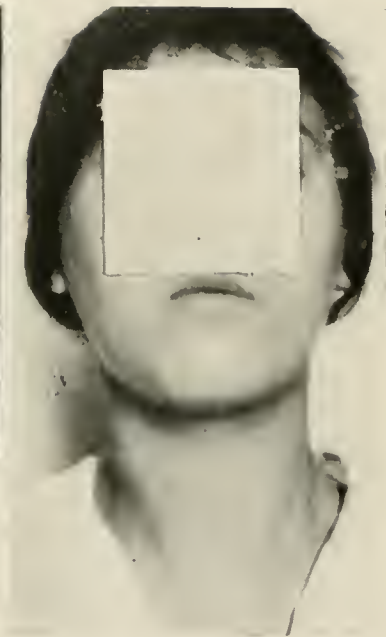


FIG. 26.

FIG. 25.—Sporadic goitre of 8 years' duration in girl of 15. Circumference of neck  $16\frac{1}{2}$  inches.

FIG. 26.—Same patient as Fig. 25. Goitre completely gone after 7 months' of treatment.



indiscretion in which the thyroid gland is forced to hypertrophy in its function of detoxication.

We may safely accept the generalization that, with few exceptions, in sporadic simple goitre the etiology is one operating on a basis of *excessive demands for thyroid hormone away from the thyroid; the organ, incapable of supplying this excess, must hypertrophy in efforts at physiological adjustment*. This process may be acute, chronic, recurrent, or intercurrent. It must be recalled that in the event of an acute causation of simple goitre, the thyroid already enlarged, may or may not resume its normal size after the disappearance of the cause—and in a considerable percentage of patients continues to grow, from mere “morbid habit of cellular proliferation” into definite tumor formation.

#### PREVENTION OF SPORADIC SIMPLE GOITRE

In the prevention of sporadic simple goitre the hereditary tendency is a strong factor to be taken into account, and prophylaxis must begin before birth of the individual. The pregnant mother who is susceptible to or already has a goitre must be under careful supervision with regard to the necessary equilibrium of the endocrine functions. This is accomplished through ample physical and mental rest, the proper dietary and personal hygiene, and the guarded administration of thyroid extract. Incidentally, thyroid opotherapy will reduce to a minimum the possibility of eclampsia.

The child born of goitrous parents must not necessarily become goitrous, though without medical supervision over a period of years, the chances to become afflicted with goitre are great. During infancy, hygiene, diet, and occasional medication are the principles of prophylaxis. In addition, the growing child should be guarded against excessive physical and mental stress and strain incident to school life. Puberty and the onset of the menstrual function constitute a prolonged period of anxiety and peril. At this time too much care cannot be given the individual with goitrous parents, for if thyroid enlargement has been avoided heretofore, the crucial test is reached, and the organ may become swollen as an incident to the first menstruation, with permanent goitre formation. The girl must have explained to her the nature of the menstrual phenomenon, and

she must be instructed in its hygiene. All physical and mental duties must be curtailed or discontinued, and the subject should stay in bed during the active period of menstruation. *This precaution taken monthly will do more than anything else in the prevention of sporadic simple goitre.* To state it more plainly, *the continuation of the usual mental and physical duties during menstruation in young girls is productive of the great majority of sporadic simple goitres seen in our midst.*<sup>2</sup>

The marital relations, pregnancy, lactation, and the menopause, occurring in persons with an inherited tendency toward goitre, likewise require dietetic and hygienic supervision, with or without thyroid opotherapy, in the interests of prophylaxis.

With regard to the prophylaxis of simple sporadic goitre resulting from the infections, little if anything, can be said. Usually the patient presents herself for the treatment of an already well-developed thyroid enlargement of varying duration; sometimes the goitre has existed for years, and the medical attendant, searching for etiological factors, discovers it to be of infectious origin either in the nature of a previous attack of an acute infectious disease, or in a coexisting pyorrhœa, tonsilitis, sinusitis, salpingitis, appendicitis, and the like. Rarely, if ever, are we called upon to *prevent* goitre originating from this source—it is its *cure* that claims our attention.

Having devoted the necessary time to the elimination of etiological factors, it may be feasible in many instances to administer a drug. While iodine, not thyroid extract, may be employed with advantage in the management of *endemic simple goitre*, *thyroid extract*, *not iodine*, serves the purpose in the sporadic type of the disease. To reverse the rule (*i. e.*, to employ thyroid extract in endemic, and iodine in sporadic goitre) may benefit a small percentage of patients, but in a greater percentage no change in the goitre will result, and in many instances the patient will become either generally indisposed, experience an increase in the size of the thyroid, or both. Though iodine is an essential ingredient of all potent thyroid products, and

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<sup>2</sup> Though the female is the usual subject of goitre, the male is not as immune as he is thought to be. In my work, devoted to goitre, I see a case of sporadic goitre in one man to every four such cases in women. In exophthalmic goitre or Graves's disease, my proportion is still greater in men; for every three women referred to me for treatment, there are approximately two men.

though Kendall's thyroxin, the most potent of thyroid substances, seems to depend upon its 60 per cent. iodine content, it is *iodine in its thyroid environment*, or *thyroid in its iodine environment*, that is required when the thyroid apparatus is to be relieved of its surplus burden of function. Thyroid *minus* iodine is impotent; *with* iodine it is thyroid as we know it—a substance at once a blessing and a curse in therapeutics and in sporadic goitre, depending upon whether it is used or abused. There is a “something” in thyroid substance, which is more or less specific in the prophylaxis of sporadic simple goitre. The nature of this “something” is still a mystery, but its action is unique and incomparable to anything else known in medicine.

#### CONCLUSIONS

1. Simple or non-toxic goitre is usually preventable.
2. Though clinically identical, endemic and sporadic goitre differ widely from an etiological and therapeutic viewpoint, and should be sharply discriminated.

## THE DANGEROUS INSANE

BY PAUL E. BOWERS, M.S., M.D.

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AMONG the insane there is a class of individuals who commit dangerous acts. They in fact commit every crime in the criminal category, as has been found by observation and study at the Indiana State Prison and the Indiana Hospital for Insane Criminals.

Of this class, Dr. Charles R. Henderson has said the following:

“There is no word here of ‘criminals’ in the legal sense. According to our laws an insane person is incapable of committing crime. But some of the insane are dangerous to society and require restraint and treatment. In many cases it is difficult to distinguish insane from criminal impulses and actions. Nervous and mental disturbances unquestionably make anti-social deeds more frequent. Therefore brief notice must here be given to this aspect of our problem.

“In the United States the conviction is gathering momentum in ever-widening circles that society is constantly in danger from certain classes of the insane, as well as from the feeble-minded and epileptic, and that measures must be taken for protection. Now that the idea of social revenge is definitely abandoned by all enlightened leaders, and the standpoint of social protection is accepted, we are in much better position to deal with the problem without passion or prejudice. It is impossible in many cases to judge whether a person is insane or not; impossible to measure the degree of responsibility or of desert, and the penalty which will satisfy abstract justice. We ought by this time to be able to free ourselves from the metaphysical confusion which disturbs the traditional penal codes built on the unverifiable assumption that a judge, a jury, or a legislature can measure the pains due to a specific act. What we can discover, with the aid of modern science, is the fact that a given person is dangerous to the life, persons or property of the community, and we can define a suitable method of education, training, medical treatment and seclusion which will, in one way or

another, protect society. All this can be done legally, with judicial safeguards of liberty, and in a way which will be best even for the person placed under treatment. Many a dangerous man has been set at liberty by courts and juries because they feared to condemn an insane person; and so individuals have been placed in jeopardy, and the trait of insanity has been transmitted to children through the vicious system based upon false assumptions."

This class of insane offenders is divided into two groups; namely, the insane criminal, and the criminal insane.

There is need for an explanation of these terms; the last term especially needs some elucidation to explain what seems to be an apparent contradiction. How can an individual be both insane and criminal at the same time? The classical school of criminologists has denied this possibility on the theoretical grounds that no crime can be committed without criminal intent, and that the insane do not have criminal intent of mind because of their insanity. From a standpoint of rhetoric this argument is logical enough. Admitting for argument's sake the plea of the classical school of criminology, that it is impossible for an individual to be both criminal and insane at the same time, we are confronted with the imperative necessity of recognizing that there is a class of violent mad-men who must be recognized as such, and restrained accordingly.

Without quibbling over hair-splitting technicalities, we are compelled to make certain practical definitions that we may have a working basis upon which to formulate our treatment of the dangerous insane.

The criminal insane individual is a person whom the court has found to be insane at the time of trial or insane at the time he committed a criminal or dangerous act. He is an individual who is positively dangerous to the welfare of society because he is unable to control his conduct by reason of mental disease or lack of mental development.

The insane criminal is an individual who has become insane or whose insanity was discovered after he was sent to prison, or an individual who becomes insane while serving sentence in prison.

In the State of New York these two classes of patients have been separated, but the distinction made between them is largely an arti-

ficial one. The criminal insane are sent to the Matteawan State Hospital, and the insane criminals are sent to the Dannemora State Hospital. Even though this arbitrary difference is made between these two groups, they are made of essentially the same types of persons. An individual who is classified as "criminal insane" may be one who has served several sentences in prison and while on parole or discharged from a penal institution he commits a crime and is found to be insane at the time of trial and because his history is unknown he is sent to a hospital for insane criminals. There is very little reason to separate these two classes since they are largely recruited from the same ranks of society and they require the same kind of treatment in the same sort of a hospital.

If we examine carefully the records of hospitals for the criminal insane, we will find that the majority of the inmates have been habitual criminals or individuals who have occupied all their lives a region that lies midway between sanity and insanity. The crimes of the insane and otherwise mentally defective prisoners show an extremely high percentage of crimes against the person. I have found that the percentage for murder among the insane prisoners was three times as high among this class as among the inmates of the prison proper. For rape, sodomy and incest, it was one and one-half times greater. Among 169 insane prisoners, 43 were murderers and 16 were convicted of assault and battery with intent to murder; 25 of them were convicted of burglary, and every burglar is a potential murderer; 11 were convicted of rape and attempt to rape, and 4 were convicted of sodomy.

The records of the Indiana State Prison of 2365 consecutive admissions show the following interesting percentages:

	Per Cent.
Murder .....	5.2
Rape, incest and sodomy .....	6.1
Murder, manslaughter, rape .....	14.6
Petit and grand larceny .....	53.2

Of 114 mentally defective prisoners:

Murder .....	16.6
Rape, incest and sodomy .....	9.8
Murder, manslaughter, rape .....	30.5
Petit and grand larceny .....	37.7

From these figures and tables we see that the majority of the crimes of the criminal insane and insane criminals is chiefly against the person. This fact is extremely pertinent and suggestive and indicates that there should be an indefinite seclusion of the individuals of these types.

The insane criminals and criminal insane suffer with the same mental and nervous diseases as do the civil insane. I have noted however no matter what type their insanities may be, their symptoms are distinctly colored with delusions of persecution; 70 per cent. of the patients at the Indiana State Hospital for Insane Criminals entertain some form of delusions of a persecutory nature; some of the delusions are organized, and some are not. In my opinion this delusional state of mind is but a reflection of the whole course of their lives and indicates the deep-seated and inherent anti-social tendencies of their mental organizations. It explains to some degree the reason why their lives have always been in conflict with society. Their delusions, when organized generally, concern society, with whom they have always been at war, but the definite fabric of their false beliefs is woven about prosecuting attorneys, judges, prison officials and the medical officers of the prison. It is a very common practice for them to prepare long statements replete with legal terms setting forth their grievances and complaints against the world at large. Their daily conversation and behavior are filled with discussions and actions which relate to crime. Very often the more intelligent insane criminals attempt to play the part of attorneys and very often plead the case of some terminal dement to the hospital officials; even the games they play in the hospital grounds are very suggestive. Here in their sports they constantly refer to, and enact, scenes of their past lives. They play at having jails and prisons; they designate one another as policemen and detectives to catch make-believe criminals who are always made the central figures of their pastimes.

In the hospitals for the criminal insane are to be found the sexual perverts of all descriptions. There were at this institution<sup>1</sup> thirty-five sexual perverts, and they constitute a very dangerous and troublesome class. It is necessary to keep them under the strictest

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<sup>1</sup> Indiana Hospital for Insane Criminals, Michigan City, Indiana.

observation to prevent them from committing homosexual acts. Quarreling and fighting among them is extremely common, and this results from their love affairs and jealousies. They form attachments for each other, indulge in hugs and personal caresses and slip into one another's beds if they have the slightest opportunity. The lovers are separated and placed in different wards and in various parts of the hospital, and yet they send love notes, trinkets and favors to their sweethearts if they get an opportunity. They have even used tin salve boxes in which to send their semen to the objects of their affection. I have also noticed that the most violent love affairs occur between the white and colored men. The negroes are usually more aggressive and take the masculine part in their acts of sodomy. Some of these patients display absolutely no shame whatsoever about their perversions. Others while apparently embarrassed make splendid promises that they will never again commit homosexual acts, but of course their promises are broken the first time they have an opportunity.

The insane criminals whether they are still in prison or in hospitals for the criminal insane are constantly manufacturing dangerous weapons. They display their ingenuity by converting the most harmless things into instruments of assault. Out of pieces of wood, spoons, tooth brushes, pens, pencils, stones and even thorns from plants, they make daggers. They steal socks, if they get an opportunity, and fill them with pebbles, sand, cinders, earth, pieces of soap or anything else they can find, with which to make "black jacks" to use in personal encounters. It is not an uncommon thing for them to make saws of clock springs. It is necessary to inspect the hospital furniture at very frequent intervals to see whether or not the reinforcing rods used in bracing chairs have been removed. In several instances the inspecting officers failed to find the missing rods. The thefts had been carefully concealed in a most novel manner. The patient, after removing the rod, molded a bolt head of putty and placed it at the site of the missing rod; wooden pins have been substituted for the same purpose. They make clubs by rolling newspapers and magazines tightly together, then soak them in water and wrap them firmly with bits of grass or string. It is necessary to search their clothing at very frequent intervals for their home-made weapons.

The criminal insane and the insane criminals do not belong in prisons or in civil hospitals for the insane; they interfere with all reformatory methods. They cannot be disciplined as are the normal prisoners; they create disturbances, are dangerous to the physical welfare of mentally normal prisoners and institutional officers. The presence of epileptics, mattoids, paranoids, paranoiacs, imbeciles and sexual perverts in our prison populations is a menace because of their dangerous tendencies and lack of capacity to adjust themselves to the environments and discipline of penal institutions. They threaten the lives of their fellow inmates and the institutional officers and not infrequently make dangerous and vicious assaults. These persons do not belong in penal institutions, which should be relieved of their presence whenever discovered. In our hospitals for the innocent insane are to be found dangerously violent persons, congenital, homosexual perverts and persons who are constitutionally immoral who do not belong in the civil hospitals since they cannot be given the proper care and restraint in such institutions.

It has been found by experience that it is very poor policy to place these dangerous insane in a separate department in ordinary civil hospitals. It has likewise been found faulty and ineffective to set apart for the care of this same class a certain part of the prison. Under the present state of affairs a hospital cannot be converted into a penal institution and neither can a penal institution be changed into a hospital. The purpose, organization and construction of a prison are diametrically opposed to those of a hospital for the insane. The spirit of these two institutions is entirely different. The official personnel of the hospital looks upon its inmates in an entirely different manner from the prison personnel where the rules are far more strict; where there are definite tasks to be accomplished, and where certain definite punishments are inflicted for the violation of discipline.

The question now arises, what is to be done with the criminal insane, since they neither belong in an ordinary hospital for the insane, nor a prison? The solution of the problem is to be found in a hospital for the criminal insane and for this definite reason hospitals for the criminal insane and insane criminals were called into existence and developed to meet the specific problem of caring for persons who were at the same time insane and criminal. Let

us make a brief review of the history of the establishment of such institutions. The English Government was the first to initiate and construct a department for the care of this class of persons, and this was done by appropriating a special department at the Bedlam Asylum, in 1786, for the reception and treatment of criminal lunatics. Bethlehem Hospital was converted to this use in 1815; another institution of similar character was opened at Dundrum, Ireland, in 1850; another at Perth, Scotland, in 1859; and the famous Broadmoor Hospital was founded in 1863; one was established in our own country in New York State in 1859, at Auburn.

In France, after an unsatisfactory attempt to care for insane criminals at Bicetre, a separate wing was built for them at the Gaillon Prison. The criminal insane in Holland were isolated in the hospital of Bosmalen. Germany established psychopathic wards in the prisons at Waldheim, Halle, Hamburg and Bruchsaal.

There are two great fundamental reasons for the establishment of these institutions. The first, which is most important, is the social defense. Society must be defended against the dangerous and anti-social acts of all classes of individuals, whether they be criminal, insane, feeble-minded, epileptic or otherwise mentally defective. The first great principle regulating our dealings with them must be that of social preservation. Our safety must be equally insured against the robber who would take our money or our life, or the dangerous paranoiac who kills in a wild, homicidal mania, reacting to the systematized delusions of persecution, or the mentally irresponsible, erratic imbecile who may murder a helpless infant merely to gratify his depraved appetites. The second reason for the existence of hospitals for the criminal insane is born of humanitarian impulses; for we recognize that the criminal, the insane, the epileptic and the feeble-minded owe their origin largely to the defects of the social organism. And since society is responsible for their existence, these defective, delinquent and dependent classes must share our humanity and our pity.

Without discussing any further in the abstract the functions of the hospital for insane criminals, its uses may be briefly explained by a short account of its practical workings. These institutions are so

comparatively few, that but a relatively small number of persons have any definite conception of them, or the valuable services they render.

The Indiana Hospital for Insane Criminals was built in the year 1910, entirely by prison labor. It is located on a plot of ground comprising four acres adjacent to the Indiana State Prison. It is surrounded by a brick wall twenty-four feet in height. The building is three stories high, built in the form of a Y. There are thirty-two private rooms on each floor. This particular model of architecture was carried out so that the supervisor on each floor could at all times have a view of two day rooms, halls, dormitories, and dining-rooms, from any front location. It is at once apparent to the visitor that the institution partakes of the character of a hospital and a prison. It is equipped with offices, a hydrotherapy, a surgery, a drug room, and a psychological laboratory. All the windows have bars of the outside basket variety. It was constructed with a view in mind to prevent escapes of the patients. The interior of the building is furnished entirely in terrazzo and this finish makes it very easy for the institution to be kept scrupulously clean.

The institution is under the same Board of Control and management as is the prison, but it is an entirely separate institution. Its medical administration is governed by a medical director who conducts its internal affairs. The attendants of this hospital are men who have had experience as prison guards and as attendants in hospitals for the insane. The wages they receive are about twice the amount of remuneration ordinarily paid this class of help in hospitals for the insane. This enables the administration to secure a better class of attendants than usual. The discipline is entirely different from that of the prison and conforms to the ordinary ideals of hospital government. Owing to the fact that the majority of the patients have served sentences in prison, their conduct in many respects is more orderly and regular than in civilian hospitals; kindness, gentleness and firmness is the triad of qualities that make for discipline. An endeavor is made to have the patients understand that they are in a hospital and not a prison. In the day rooms they are allowed books from the library; they have games and musical instruments for their entertainment. They are afforded recreation

outside the building in the form of ordinary field sports, as baseball, basketball and intensive gardening.

Within the hospital grounds there has been constructed a weaving shop which contains many hand looms and sock machines. In this department blankets, sheeting, ticking, shirting, toweling, rugs, etc., are manufactured. Occupational therapy has proved very valuable in the treatment of the dangerous insane.

The following is an abstract of the laws which relate to the purpose and function of the Indiana Hospital for Insane Criminals:

#### CHAPTER 87, ACTS 1909, INDIANA

##### *Lunacy Commission.*

SECTION 8. Whenever the physician of the Indiana Reformatory or the physician of the Indiana State Prison shall certify to the general superintendent or warden that any convict therein is insane, giving in full the signs, symptoms and conditions in detail on which his diagnosis is based, together with the full history of said convict as far as obtainable, the general superintendent or warden shall then, if fully satisfied of the insanity of such convict, report such case to the governor of the state transmitting with such report a complete transcript of all of the papers filed in the case, which transcript shall be signed by the general superintendent or warden and sealed with the seal of the institution. Upon the receipt of such transcript, the governor shall direct the general superintendent or warden to convene a lunacy commission to be composed of two physicians and one justice of the peace, resident in the county in which such reformatory or prison is located, but having no connection with such institution, whose duty it shall be to investigate and examine into the mental condition of the person alleged to be insane; and after such investigation and examination to report in detail and in writing his condition to the general superintendent or warden, who shall upon receipt of said report, transmit it to the governor: PROVIDED, that at least five days prior to the holding of such examination by said lunacy commission, it shall be the duty of the general superintendent or warden to notify, in writing by United States mail, the next friend or nearest relative of such convict alleged to be insane, if the records of the institution contain the address of such friend or nearest relative or if the same can be ascertained, of the intention to hold such examination and of the time and place where the same will be held and that such friend or relative may be present in person or by representative if he so desire, and the secretary of the state board of charities shall also be notified of such examination.

##### *Report—Governor's Order.*

SECTION 9. Upon receipt of the report of said lunacy commission by the governor, he shall, if fully satisfied of the insanity of such convict and as to the wisdom and justice of the proposed transfer, issue an order committing said convict to the Indiana Hospital for Insane Criminals and commanding the general superintendent or warden to transfer such insane convict to the Indiana Hospital for Insane Criminals, the order for such transfer to accompany the

convict and to become a part of the records of his case and to be kept on file as other records are kept.

*Recovery of Sanity.*

SECTION 10. Whenever any convict, who shall have been confined as insane in the Indiana Hospital for Insane Criminals, shall recover his sanity before the expiration of his sentence or before the expiration of the maximum limit of an indeterminate sentence, the warden and physician in charge shall so certify to the governor, who, having fully satisfied himself as to the fact of sanity, shall thereupon order such convict to be transferred to the penal institution from which he was removed to the insane hospital.

*Term of Confinement.*

SECTION 11. Whenever the insanity of any convict confined as insane in the Indiana Hospital for Insane Criminals continues beyond the expiration of his sentence, or beyond the maximum limit of his indeterminate sentence, he shall be kept in such hospital so long as his insanity continues and in case any such convict shall recover his sanity after the expiration of his sentence or after the expiration of the maximum limit of his indeterminate sentence, the warden and physician in charge shall certify such recovery to the governor, who, having fully satisfied himself as to the fact of such sanity, shall forthwith order his discharge by the warden, which discharge shall be immediately reported by the warden to the institution from which such convict was transferred and to the secretary of the state board of charities.

*Criminal Insane in State Hospitals.*

SECTION 12. Insane convicts, sentenced to the Indiana Reformatory or to the Indiana State Prison and confined in any of the state hospitals for insane at the time of the passage of this act, shall be transferred to the Indiana Hospital for Insane Criminals, as soon as practicable after the issuance of notice by the governor of the completion and readiness of such hospital to receive patients. In every instance the transcript of the record of the original inquest and of any intermediate proceedings must accompany the patient. The expenses of such transfer shall be borne by the institution from which such patient is removed.

*Credit on Sentence.*

SECTION 14. An insane convict shall receive credit on his sentence for the time while he is insane and under treatment in the Indiana Hospital for Insane Criminals to the same extent as if he were confined during that time in the institution to which he was originally sentenced.

*Insanity Defense—Sentence.*

SECTION 16½. After the passage of this act, if upon the trial of any male person accused of a felony the defense of insanity is interposed whether upon a special plea or a general plea of not guilty, the court or jury trying said cause shall make a finding both as to the sanity of said defendant at the time so claimed and as to whether he committed the act as charged. And if it shall be found in favor of said defendant on such plea of insanity but against him as to the commission of the act as charged, he shall, upon order of the court, be committed to and confined in the Indiana colony for the insane criminals in like manner and on such conditions and for such terms as is now provided for by law for the confinement of insane criminals in a state hospital for the insane.

## CHAPTER 298, ACTS 1913, INDIANA

*Insanity—Criminal Cases—Plea of Insanity.*

SECTION 1. Be it enacted by the general assembly of the State of Indiana, That when the defendant in a criminal cause desires to plead that he was of unsound mind at the time the offense charged was committed, he himself, or his counsel, must set up such a defense specially in writing, and the prosecuting attorney may reply thereto by a general denial in writing.

*Evidence.*

SECTION 2. At the trial of such cause evidence may be introduced to prove the defendant's present sanity or insanity.

*Time of Insanity—Duty of Court and Jury.*

SECTION 3. In all cases where a plea of insanity is interposed as a defense it shall be the duty of the jury or the court if tried by it if the defendant is found not guilty, to find, and the jury or court shall be required to find, whether the defendant committed the act charged in the indictment or the affidavit, and if so, whether the defendant was sane or insane at the time of the commission of the act, and whether not guilty because he was insane at the time of the commission of the act.

*Commitment of Criminal Insane.*

SECTION 4. If the court or jury trying the cause find the defendant not guilty on the ground of insanity, the court shall find as to the defendant's sanity at the time of the trial, and if the court shall find that the defendant is insane at the time of the trial, he shall order the defendant, if a male person, to be committed to the Indiana colony for the criminal insane, and, if a female person, to be committed to any hospital of the state where the female insane are confined; or if he shall find that the defendant is sane at the time of trial, but the recurrence of such an attack of insanity is highly probable, he shall order the defendant to be committed as above provided. Such person shall be confined therein until released as hereinafter provided.

*Discharge from Custody.*

SECTION 5. At any time after six months from the date of said commitment, any person so confined in a hospital for the insane, may file his or her application to be discharged, in the court from which they were committed, and, upon satisfactory proof being made to such court of the restoration of the sanity of such person and that the recurrence of such an attack of insanity is improbable, the court shall order his or her discharge from such institution, and enter a final judgment discharging him or her; PROVIDED, HOWEVER, That a second or subsequent applicant for discharge shall not be made within two years from the time of any previous application.

*Commitment Before Trial.*

SECTION 6. That when at any time before the trial of any criminal cause, or during the progress thereof and before the final submission of the cause to the court or jury trying the same, the court, either from his own knowledge, or upon the suggestion of any person, has reasonable ground for believing the defendant to be insane, he shall immediately fix a time for a hearing to determine the question of the defendant's sanity and shall appoint two competent disinterested physicians who shall examine the defendant upon the question of

his sanity and testify concerning the same at the hearing. At the hearing, other evidence may be introduced to prove the defendant's sanity or insanity. If the court shall find that the defendant has comprehension sufficient to understand the nature of the criminal action against him and the proceedings thereon and to make his defense, the trial shall not be delayed or continued on the ground of the alleged insanity of the defendant. If the court shall find that the defendant has not comprehension sufficient to understand the proceedings and make his defense he shall commit the defendant to the Indiana colony for the criminal insane, or, if a female, shall be committed to any hospital of the state where the female insane are confined. Whenever the defendant shall become sane, the superintendent of the insane hospital shall certify the fact to the proper court, who shall enter an order on his record directing the sheriff to return the defendant, or the court may enter such order in the first instance whenever he shall be sufficiently advised of the defendant's restoration to sanity. Upon the release of any defendant so committed he or she shall then be placed upon trial for the criminal offense the same as if no delay or postponement had occurred by reason of defendant's insanity.

It is at once recognized that these laws are in some respects quite faulty, and attempts have been made, and future efforts will be made, to have these statutes so improved as to meet the standards which have been attained in medico-legal science.

The presence of a hospital for the criminal insane in the state of Indiana has had a very marked effect upon the behavior of the prisoners in the state prison. The belief has become current among many of the prisoners that very frequent infractions of prison discipline are looked upon by the prison officers as indications of mental defectiveness and they soon learn that insanity means a transfer to the hospital for insane criminals, and once they are there, their stay is indefinite. The inmates of the insane hospital are always anxious to return to prison, for in such an institution they know that they will be discharged from custody when their sentences have expired and that this is not the case in the hospital. There has also been a great reduction in the number of trial cases in the Indiana courts in which the plea of insanity has been interposed as a defense. As the result of the presence of the Indiana Hospital for Insane Criminals, insanity pleas for the defense of crime have become far less popular in Indiana, and malingering of insanity in the Indiana State Prison has been reduced to practically nothing.

Just as soon as psychopathic laboratories become an integral part of the legal machinery of our courts of justice, those individuals

who commit dangerous acts because of unsoundness of mind will be promptly discovered without running the whole gamut of criminal court procedure, as it is now practiced. When those individuals are discovered, they will be sent, without further loss of time, to hospitals for the criminal insane, and they will not be turned loose upon society because they are "not guilty of crime, because they are insane," and they will not be sent to ordinary prisons as normally minded felons, to be punished for acts which were purely symptomatic expressions of their unrecognized disorders. The psychopathic laboratory in the prison will at once discover and classify the dangerous insane, who find their way into our prisons, because of the miscarriage of justice, and those prisoners who became insane while serving sentence, and are therefore dangerous to the rest of the prison inmates, will be transferred to hospitals for the criminal insane. Two very definite conclusions are to be drawn from the study of criminal psychiatry; the modern psychiatrist making due allowances for rhetorical differences, academic and legal finesse, recognizes that there is a distinct class of dangerous or criminal insane, who are to be discovered by psychopathic laboratories in the criminal courts, and in penal institutions. Since there does exist a dangerous mentally defective class, it becomes necessary for the states of the union to establish hospitals for the criminal insane, or to make equivalent provision for the care of these individuals in connection with other state institutions.

# A CLINICAL STUDY OF MALIGNANT TUMORS OF THE ANTERIOR MEDIASTINUM AND THYMIC REGION

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## TOPOGRAPHICAL ANATOMY

I FEEL that it is useful to briefly refer to the most important points of the topographical anatomy of the mediastinal region before taking up the subject of the symptomatology of tumors of the mediastinum.

The *mediastinum* is an irregularly shaped cavity filled by numerous organs which in the thorax occupy the space comprised between the sternum spine and internal surface of the lungs.

After slightly coming in contact with the sternum, the pleura spreads out and in this spreading, which extends from the posterior aspect of the sternum to the pedicle of the lungs, it first circumscribes a space called by some the *anterior mediastinum*. Then continuing its expansion from the pedicle of the lungs to the anterior aspect of the spine, the pleura circumscribes a second space which, in opposition to the first, has been given the name of *posterior mediastinum*.

The anterior portion of the mediastinum—the most important from the viewpoint of this paper—measures in height that of the anterior vertical diameter of the thorax. In shape it may be compared to a pyramid with three sides, two lateral and one posterior. The two antero-lateral surfaces are formed by the layers of pleura which in front are attached to the sternum. They are related, especially on the right side, to the lung. The posterior surface, which is less broad, is related below to the œsophagus and thoracic aorta. It is in this almost triangular space that the following organs are contained:

The *pericardium*, which extends vertically from the xiphoid cartilage to the middle of the first piece of the sternum and horizontally to the level of the fourth intercostal space 8 to 10 centimetres to the left, and from 2 to 3 centimetres to the right of the median line;

The *heart*, whose apex corresponds with the sixth rib and at about 10 centimetres to the left of the median line;

The *arch of the aorta* which corresponds with the middle and upper part of the sternum and contains the cardiac plexus in its concavity.

On the same plane as this vessel are to be found: On the right, the *brachiocephalic trunk* and the *superior vena cava*; on the left, the common carotid and subclavian and, more externally, the pneumogastric, recurrent and phrenic nerves.

Behind these organs lies the pedicle of the lungs formed: By the *bronchi*, whose situation in the mediastinum is in the same plane as the internal portion of the second intercostal spaces; the *pulmonary arteries*; the *pulmonary veins*.

The posterior portion of the mediastinum—*posterior mediastinum*—likewise very important on account of the organs going through it, is about the same height as that of the dorsal portion of the spine. In shape it is that of a pyramid with four sides and with its apex directed downward.

The lateral surfaces are formed by the pleura which spreads apart above to receive the subclavian arteries. The posterior surface is in proximity with the spine, while the anterior surface is limited by the bifurcation of the trachea in its upper quarter, and by the pericardium in its lower three-quarters. In this irregularly quadrangular space the following organs are found:

The *thoracic aorta* and *œsophagus*, at first situated in the same transversal plane and approaching each other as they descend in such a way that the *œsophagus* ends by becoming placed in front of the aorta and contributes to the formation of the apex of the pyramid;

The *great azygos vein*, which occupies the right side of the spine behind the *œsophagus*;

The *small azygos vein*, on the left of the spine behind the aorta;

The *thoracic duct* lying between the two azygos veins.

Finally, the connective tissue, lymph-nodes, branches of the great sympathetic and pneumogastric nerves which surround the *œsophagus*.

The lymph-nodes of the mediastinum receive the lymphatic vessels of the pleura, lungs, trachea, bronchia, heart, pericardium, and

thoracic walls. According to Baréty, the groups of lymph-nodes deserving special attention are: (1) The peritracheo-bronchial, both right and left; (2) the right and left sub-bronchial, and (3) the interbronchial groups.

#### MORBID ANATOMY

*Origin of primary malignant tumors of the mediastinum. Tumors of the thymus.*

The primary malignant tumors of the anterior mediastinum have as starting-points the various tissues and organs belonging to the mediastinum. In a schematic way they are: The cellular tissue of the mediastinum, an exceptional, perhaps even doubtful, starting-point; the regional lymph-nodes, bronchial lymph-nodes, lymph-nodes of the hilum or of the mediastinum itself, and lastly, the thymus or its vestiges.

It is generally admitted that the cellular tissue of the mediastinum may give rise to true sarcomatous growths—round cell, fusiform cell or mixed neoplasms—and that the lymph-nodes give rise to *lymphosarcomatous* tumors of the anterior mediastinum, whose histologic structure—reticulum and lymphoid cells—recall the lymphatic origin of the growth.

The thymus itself, or its vestiges, when persisting in an abnormal way behind the sternum, is often considered as an important starting-point of primary malignant tumors of the mediastinum and this origin has been for a long time admitted by a large number of pathologists.

The question of the thymic origin of mediastinal growths has nevertheless given rise to much controversy. The supposition that malignant neoplasms of the mediastinum derived from the thymus itself has been sharply denied by several authorities, especially by Hoffmann, who examined and criticized most of the cases that were known up to 1896, and by Lohrlich, who studied the cases reported between 1896 and 1901, and lastly by Nicol.

It is impossible for me to enter into the details of the controversies that have arisen on various aspects of this subject. It is sufficient to mention the general data which have been obtained as a

result of the numerous discussions concerning the possible origin of mediastinal tumors.

If, on the other hand, among the organs of the mediastinum, the thymus can and even should be regarded as a frequent starting-point of malignant mediastinal tumors—given especially the anatomical position of this organ, its phenomena of evolution, the abnormal persistency of certain of its elements, as well as the seat, histologic structure and shape of certain growths of the mediastinum—it is, on the other hand, without any really valid proofs that a large number of mediastinal growths have been and still are considered as tumors of thymic origin. Such is the opinion of Nicol, Simmonds and several other observers.

Nicol even mentions Hoffmann's opinion, who goes farther than the majority of other pathologists, and concludes from his large statistics that there are no "sure" signs of the thymic origin of primary malignant mediastinal neoplasms.

An opinion such as this is clearly too absolute. In point of fact there exists in the literature a long series of cases of mediastinal tumors in which characteristic thymic elements—especially Hassal's corpuscles—were unquestionably recognized and demonstrated.

In this respect the studies of Steudener, Bramwell, Hahn and Thomas, Grandhomme and Wiesel—quoted by Nicol—should be mentioned. Nevertheless, in a large number of very complete examinations of mediastinal tumors, with autopsy and histologic examinations comprising pieces of tumors quite as different as possible, typical elements of the thymus have most usually not been detected.

The proper attitude to assume for the present seems to me to prudently leave the question open and to take into account only those tumors of the thymus that are indubitably proved such, although such instances are rare.

#### MORPHOLOGY AND STRUCTURE OF MALIGNANT TUMORS OF THE MEDIASTINUM

Primary malignant tumors of the mediastinum, as I have said, are sarcomata, lymphosarcomata and carcinomata, and at autopsy are frequently found in the shape of heavy, massive neoplasms,

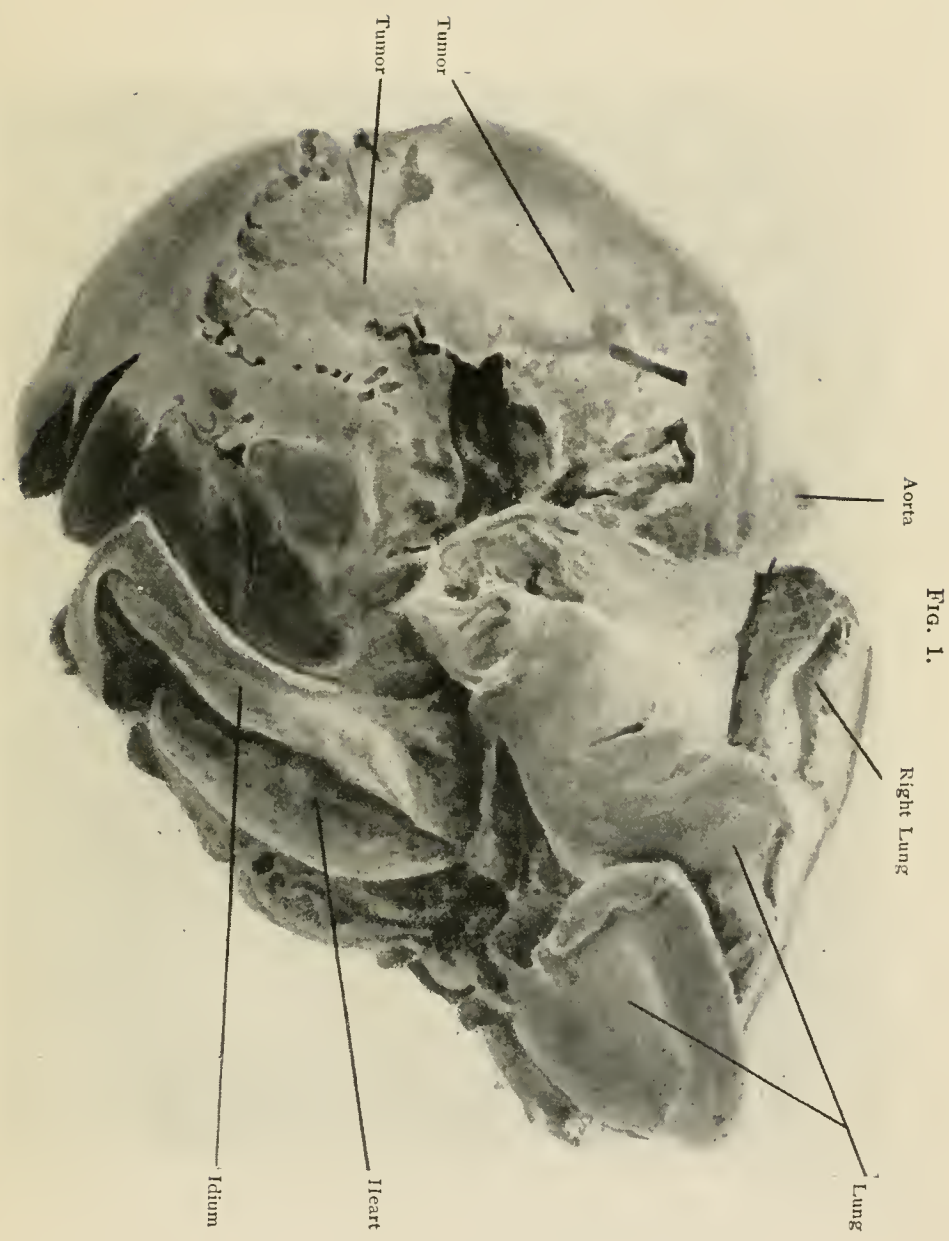


FIG. 2.

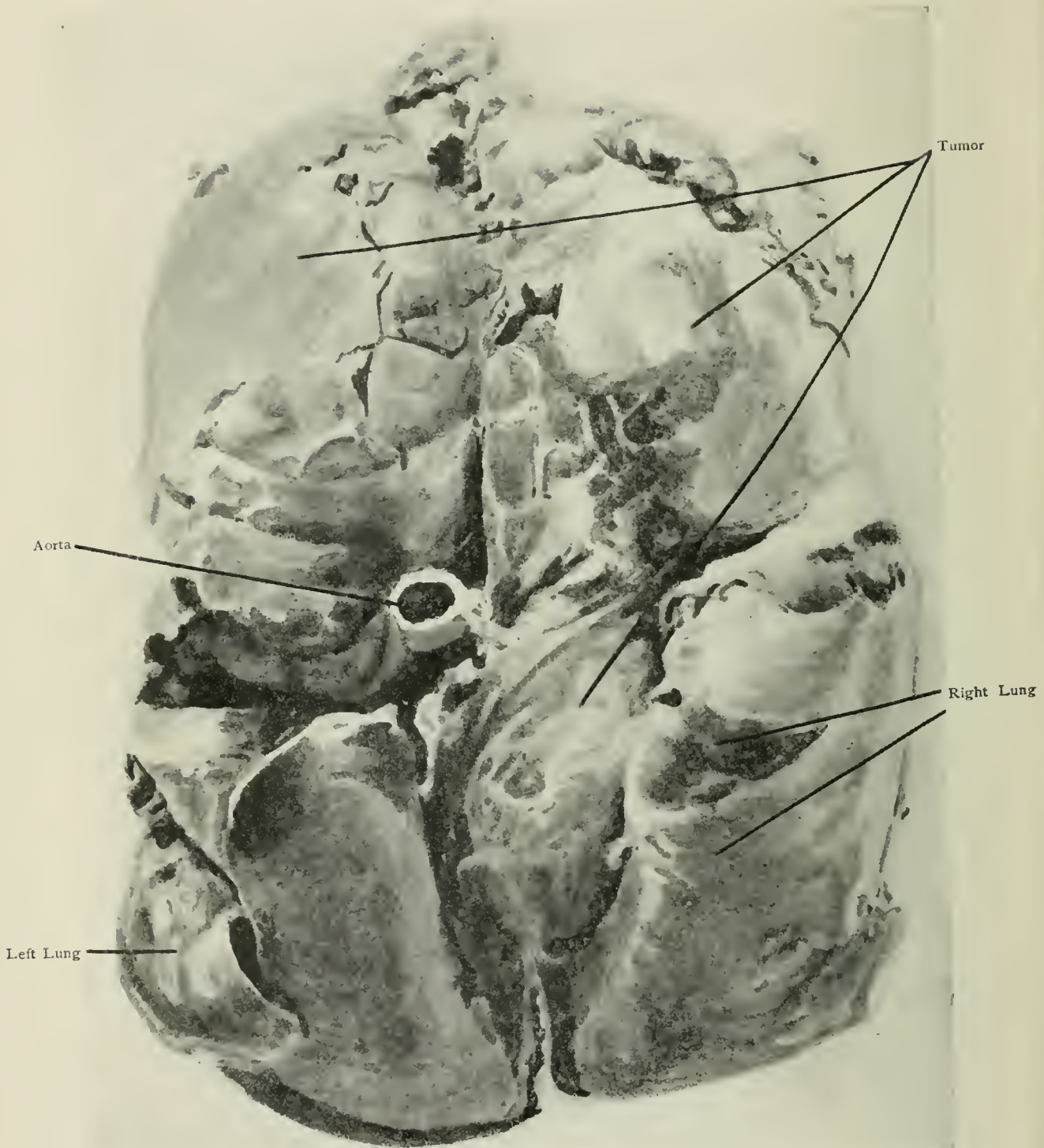


FIG. 3.

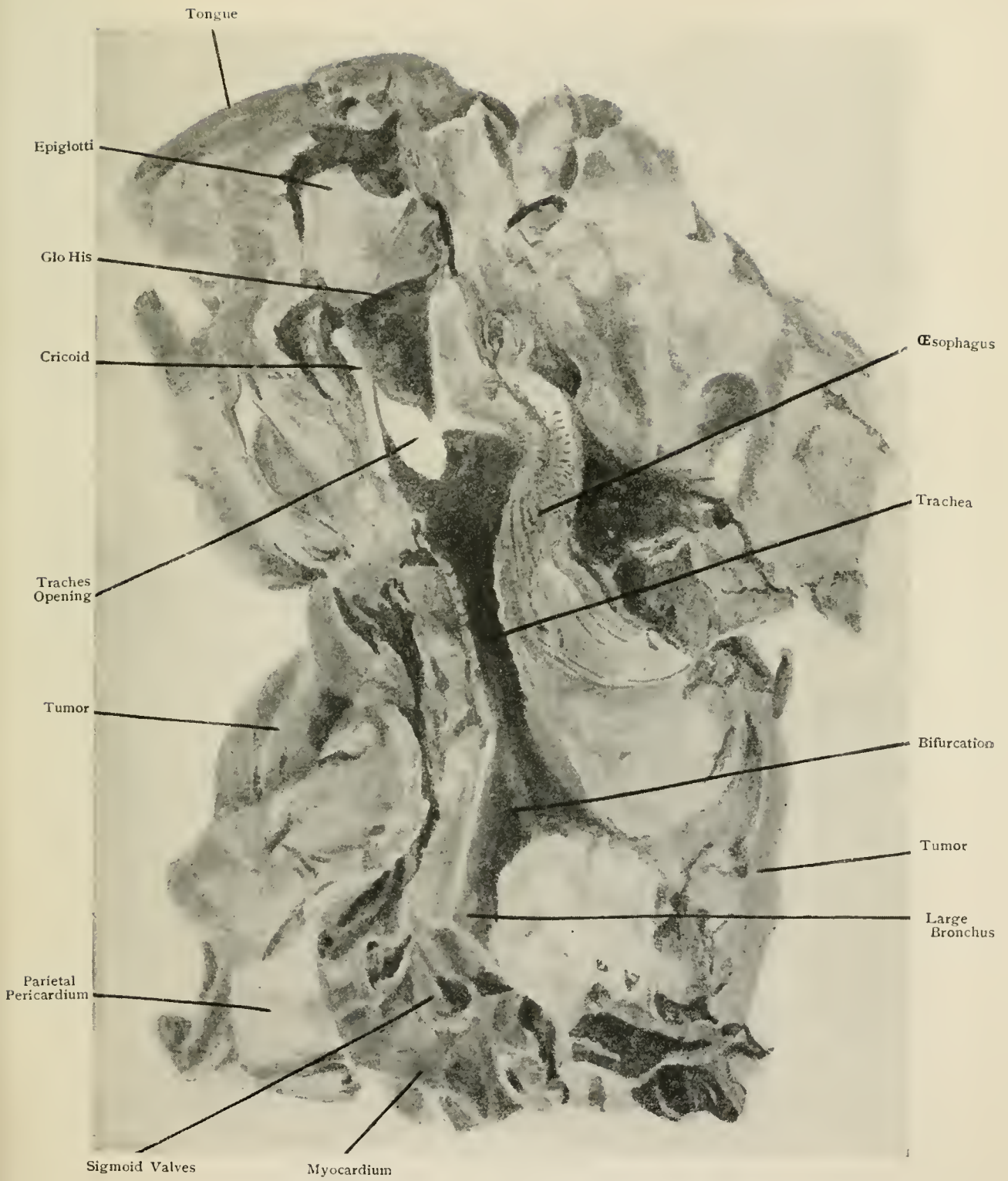
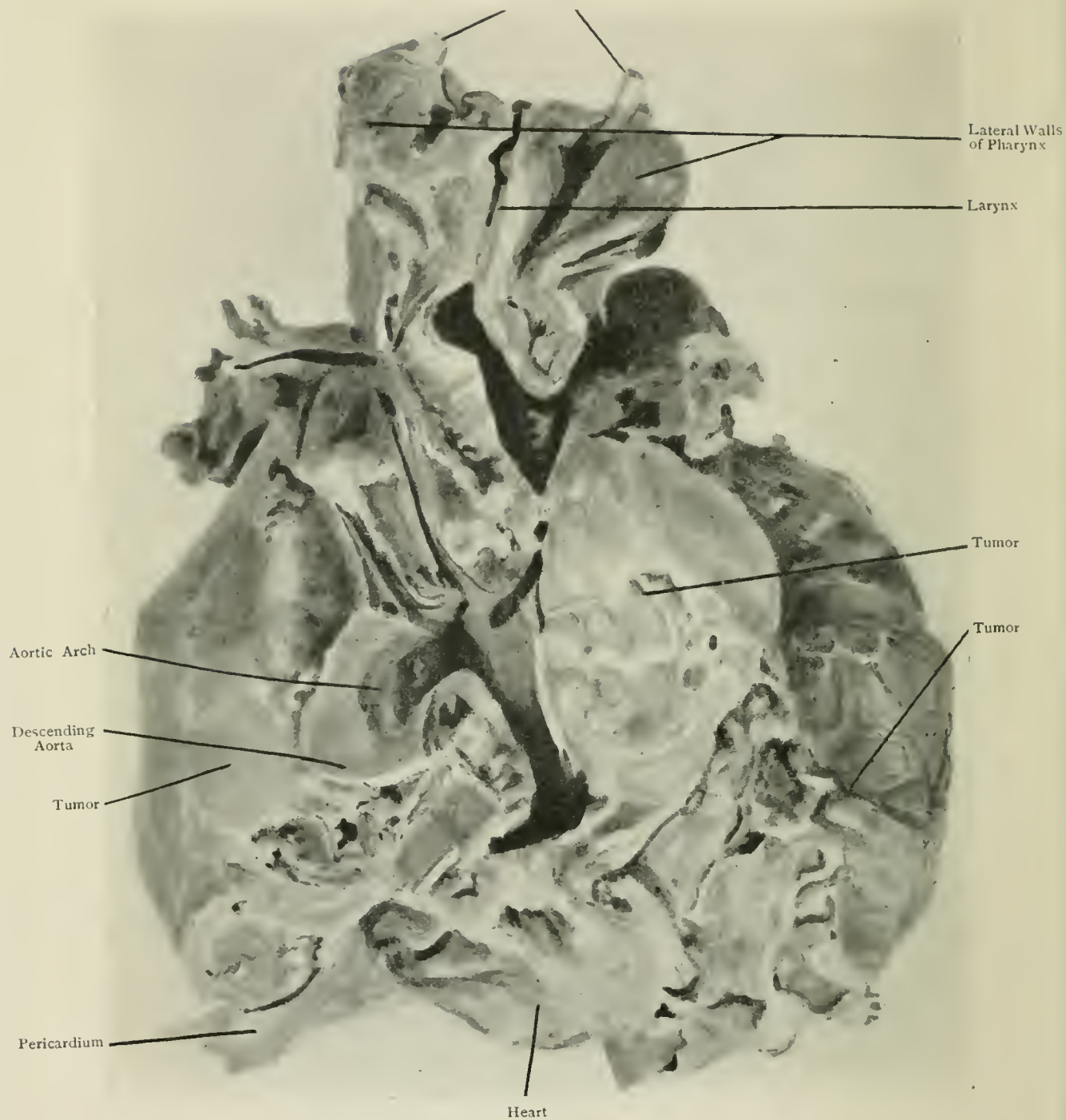


FIG. 4.  
Horns of the  
Ligoid



occasionally very large in size, filling the entire anterior and superior portion of the thorax, spreading apart and pushing back the free edges of the lungs, covering the pericardium and heart and occasionally embedding the organs contained in the mediastinum, as can be seen in Figs. 1, 2, 3 and 4.

The organs most commonly pushed aside or embedded in the growth, and afterwards compressed or infiltrated by these neoplasms are: The arteries and veins of the region—ascending aorta, aortic arch, arterial trunks given off by the arch of the aorta, the brachiocephalic veins, the pulmonary arteries and veins, the upper vena cava, the great azygos vein and the bronchial arteries and veins, the nerves passing through the mediastinum—pneumogastric, recurrent, phrenic and sympathetic—the trachea and bronchi, the œsophagus, heart and lastly the lungs and pleura.

Mediastinal neoplasms usually assume the shape of irregularly rounded globular masses; their surface may be even and smooth or, on the contrary, irregular and bossed, offering a more or less lobular aspect. They are frequently covered by a fibrous, rather dense and tense membrane which distinctly separates them from the adjacent structures.

Large neoplastic nodules, resembling large infiltrated lymph-nodes, are frequently found adherent to the principal tumor mass.

The consistency of these growths varies, but usually it is firm and hard, less commonly soft. In color they vary from a pearly white to a grayish or pinkish white. Often fine hemorrhagic spots, accompanied or not by brownish foci of softening, are seen over a portion or the entire surface when the tumor is incised.

On section these neoplasms usually offer a smooth, glistening surface, showing a fibrous or finely fibrillated structure having a more or less distinct lobulation.

*Microscopically*, the histologic structure of malignant mediastinal growths appears in the well-known shape of sarcomata—round or fusiform celled or mixed types—lymphosarcomata and carcinomata.

I cannot here enter upon the minute structure of these tumors, which can be found in any text-book on general pathology and which is still a very much discussed subject. I shall merely recall that in the majority of cases and in spite of the most carefully conducted

researches, the microscope—usually sufficient for making a diagnosis of the nature of most neoplasms—as yet does not furnish a solution of the problem as to the exact starting-point—the thymic origin in particular—of malignant growths of the mediastinum.

In point of fact there are few cases of mediastinal tumor in which the characteristic histologic elements of the thymus have been detected and in which the microscope has revealed Hassal's concentric corpuscles or cells derived from the cortical or medullary strata of the thymus.

Hence it can be concluded that malignant tumors of the anterior mediastinum are described too often and without sufficient control as neoplasms of the thymus. One should be extremely reserved in affirming their thymic origin and only to admit their relationship to the thymus when the characteristic glandular elements have been proved to be present in the tumor.

#### CLASSIFICATION AND NOMENCLATURE

The question of classification and nomenclature of primary malignant tumors of the anterior mediastinum is a moot one, and although a lengthy discussion is impossible for want of space, I cannot, however, pass over the recent researches of Simmonds and Nicol, who have each attempted to establish a classification of mediastinal growths which might fulfil the requirements of the more recently recorded cases.

Both these observers, basing themselves on a certain number of personal cases of mediastinal growths, very exactly examined and severely criticized, have each presented an attempted classification to which I shall refer.

The question of classification is a complex one from the fact that many observers have considered and still do consider, without any valid reason, that the mediastinal growths that have come under their observation are undoubtedly tumors of the thymus.

Others have abused the convenient term of "lymphosarcoma of the anterior mediastinum," and without proper control have applied it to too many cases.

Finally, the positive thymic origin of a large number of primary malignant tumors of the mediastinum remains, in spite of all patho-

logic researches, impossible to establish and the exact interpretation of the normal elements of the thymus itself—especially its round cells, authentic lymphocytes for some, modified epithelium according to others—is still a matter of controversy.

Simmonds divides tumors of the thymus into three groups according to the origin of the neoplastic cells, that is to say, according to the part of the gland from which they are derived.

The *thymomes* arise from the round cells of the cortex of the gland; the *sarcomata* are derived from the cells of the interlobular connective tissue, while *carcinomata* develop from the medullary strata.

Nicol's classification and nomenclature are somewhat different and I will give them in a tabulated form:

I. MALIGNANT TUMORS OF THE THYMUS (Tumors having demonstrable thymic origin).

(a) *Tumors originating from the round cells of the thymus.* Lymphosarcomata and carcinomata of the thymus (carcinomata with cells of the cortex, according to the interpretation given to the round cells).

(b) *Tumors originating from the interlobular connective tissue.* Fibrosarcoma of the thymus.

(c) *Tumors originating from the medullary epithelial cells.* Carcinomata of the thymus (medullary cell carcinoma).

II. LYMPHOSARCOMATA OF THE MEDIASTINUM. Tumors having a demonstrable lymph-node origin.

III. TRUE SARCOMATA OF THE MEDIASTINUM. Tumors originating from the mediastinal connective tissue—round or spindle cell sarcoma, etc.

#### ETIOLOGY

The etiology of primary malignant tumors of the anterior mediastinum is still absolutely obscure. None of the causes that have so far been invoked for explaining the origin of these neoplasms have been found satisfactory. Some observers have maintained that traumata and heredity might play a certain part in the development of mediastinal growths. But in reality the existence of these factors has never been proved excepting in a very small number of cases, so that their influence is far from having been distinctly demonstrated.

## FREQUENCY IN RELATION TO AGE AND SEX

Males are slightly more prone to primary malignant mediastinal growths than females. In Riegel's statistics the number of males, as compared with females, is 2.4 to 1.1.

As to the age of the subjects, the affection is rare in childhood and infancy and appears to preferably develop in young adults. Bollag (*Diss.*, Zurich, 1887), out of a total of one hundred cases collected from the literature, only found nine subjects under the age of fifteen years. Pless (*Diss.*, Griefswald, 1867), in statistics comprising eighty-five cases of malignant tumors of the anterior mediastinum, found no instance in childhood. Eger (*Diss.*, Breslau, 1872), out of a total of fifty-five cases, only found one patient under the age of ten years, while Eichhorst found four patients under ten years of age out of a total of thirty-five cases.

As far as I am concerned, the six cases that I here publish are three males and three females, the patients' ages being respectively five years and six months, twelve years, sixteen years, two twenty-seven years and the last thirty-eight years.

## SYMPTOMATOLOGY

In his paper "Des tumeurs malignes du médiastin antérieur," H. Rendu briefly outlines the symptoms offered by mediastinal growths and he says: "There are circumstances in which the idea of an intrathoracic tumor imposes itself of necessity on the mind of the physician.

"When in a subject not predisposed by diesthetic antecedents, a cough develops, likewise difficult breathing, although no changes in the lung or the heart can be detected by auscultation, this is already a presumption that the mediastinum is involved.

"The certitude becomes almost absolute when to these symptoms engorgement of the veins of the neck, cyanosis, œdema of the face and upper limbs become added, and above all the appearance of lymphatic tumors in the supra-clavicular region manifestly belonging to productions of the same nature developed within the thoracic cavity. . . . .

"But such a complete collection of symptoms is not always met with and on the other hand when it exists with these characters the process will have almost reached its ultimate phase.

"Therefore it is necessary to search for circumstances which, at the onset of the affection, may put the clinician on the road to a diagnosis."

The study of well-taken case histories giving sufficient details in cases of malignant tumors of the mediastinum leads me to about the same conclusions as Rendu.

#### SYMPTOMS OF THE ONSET

Although a certain number of perfectly characteristic symptoms appear in the advanced phases of malignant growths of the mediastinum, it is, on the other hand, generally impossible in the early phase of these tumors to detect signs of their onset which offer any real value.

From this viewpoint I have particularly examined the six cases here reported in relation to the early symptoms presented by the patients. It has been quite impossible to discover any distinct constant characteristic signs present at the onset of development of mediastinal neoplasms. The case histories of the six patients reveal as early phenomena, only rather indefinite symptoms represented by more or less marked functional disturbances of the respiratory and circulatory systems. At first, evidences of an intrathoracic obstacle are vague, often only represented by cough. But they soon become more marked, causing progressively increasing distress.

The cough, instead of subsiding, persists, becoming paroxysmal, occasionally similar to pertussis. Slowly and progressively dyspnoea ensues and becomes marked. The pulse-rate increases, cardiac palpitations arise and occasionally a slight cyanosis of the lips and face develops relatively early in the process, while fleeting œdema of the lids, neck and upper part of the thorax supervene.

All these symptoms have nothing particularly characteristic and are usually insufficient for making an early diagnosis of malignant tumors of the mediastinum. In the majority of cases these symptoms are interpreted as indices of the onset of some bronchopulmonary, pleuropulmonary or cardiac affection, and usually neither the patient nor physician pays much attention to them.

If sometimes the early diagnosis of malignant mediastinal tumor has been made this is due to the fact that the development of the intrathoracic neoplasm is, in reality, in a more advanced phase than

was at first supposed. In point of fact, in some instances, certain symptoms properly belonging to fully developed mediastinal growths already existed besides the mild functional symptoms referred to.

The so-called *retro-sternal* pain, considered as a symptom, especially an early symptom of a tumor of the mediastinum, does not appear to possess the diagnostic value that some have attributed to it. Seated behind the sternum, usually at the level of the manubrium, this dull constrictive pain, without irradiations, has been mentioned by Kaulich, Rendu and others as having a real importance from the viewpoint of diagnosis.

According to these observers the retro-sternal pain usually precedes all the other symptoms and Kaulich states that in one case it was the only symptom present.

I feel that this question is as yet a moot one as the majority of cases so far published do not appear to justify the opinion of Kaulich and Rendu. In my six cases, I was able to note the complete absence of retro-sternal pain as a constant and single symptom of the onset of the tumors in question. It would therefore seem logical to conclude that primary malignant tumors of the mediastinum are, in general, deprived of any constant characteristic symptom at their onset.

*Symptoms belonging to the phase of full development of mediastinal tumors. The mediastinal syndrome.*

It is not my intention in this short survey to enter into the details of each of the physical signs characterizing malignant mediastinal growths, or to discuss all the symptoms belonging to the semeiologic group created by Dieulafoy under the name of the *mediastinal syndrome*. For that matter, it would be quite superfluous to describe well-known symptoms, therefore I shall merely recall the principal ones. Very briefly summarized these are: Enlarged supra-clavicular and cervical lymph-nodes, deformity of the thorax, forward bulging of the sternum, various changes in pulmonary percussion and auscultation, such as sternal dullness, compression souffles, etc. Also more or less intense circulatory disturbances, especially in the domain of the superior vena cava.

In reality, most of these symptoms are merely signs of compression of the intrathoracic viscera. In fact, the most intimate rela-

tionship unites the mediastinal organs such as the heart with the large vessels, the trachea and large bronchi, the œsophagus, the pneumogastric, phrenic, splanchnic and recurrent nerves, the great azygos vein, thoracic duct and thoracic lymph-nodes (Dieulafoy).

Consequently it is difficult to conceive how a tumor developing in so important a region as the mediastinum can progress without sooner or later giving rise to serious disturbances.

When the trachea becomes pushed aside or flattened, or its calibre reduced, marked symptoms such as dyspnœa, etc., ensue. When the œsophagus becomes deviated, flattened or its lumen narrowed, dysphagia results, solid food will not pass, while fluids are ingested with difficulty. If the great azygos vein becomes compressed, since it receives the small azygos and seven or eight of the right intercostal veins, stasis results and a collateral circulation develops made manifest by a network of distended veins in the thoracic region. If the pneumogastric becomes involved paroxysms of coughing ensue with severe attacks of suffocation, while the recurrent nerve when involved will give rise to laryngeal disturbances, hoarseness, dysphonia and spasm of the glottis.

"It is this *ensemble* of symptoms which constitutes the *mediastinal syndrome* which, when more or less complete, will allow one to affirm the existence of a tumor of the mediastinum" (Dieulafoy).

#### DIAGNOSIS

The diagnosis of malignant growths of the mediastinum is relatively easy to make when the ensemble of the symptoms mentioned exists or when, at least, the principal signs of an intrathoracic growth are present.

On the contrary, the diagnosis becomes extraordinarily difficult, sometimes even impossible, when, as in certain instances of tumors at their onset, the symptoms are almost completely wanting or assume characters common with those of other affections of the lungs, bronchi or heart.

It is essential to insist upon the fact that the majority of mediastinal neoplasms present a more or less long phase during which an exact diagnosis can only be made with great difficulty for lack of distinct signs.

Therefore, malignant tumors of the mediastinum generally begin without any distinctive symptoms and it is only after a variable length of time that they manifest their presence by some one of the symptoms mentioned.

#### DIFFERENTIAL DIAGNOSIS

Very different affections of the mediastinum may, in fact, offer absolutely identical symptoms with those of a neoplasm, hence the diagnosis of tumor often remains undecided for some time as in Cases I, II, V and VI.

The differential diagnosis of mediastinal tumors must be made with the following affections: *Primary or secondary cancerous adenopathies of the mediastinal or thymic lymph-nodes; tuberculous adenopathies; syphilitic mediastinitis or mediastinal syphilomata; abscess of the mediastinum; aortic aneurysm; retro-sternal goitre; mediastinal pleurisy; and lastly, dermoid or hydatid cysts.*

*Cancerous Adenopathies.*—All that is necessary is to recall that cancer of the mediastinum has an insidious development; after a latent phase of indeterminate duration, it manifests its presence by definite symptoms especially belonging to the mediastinal syndrome. Its progress is usually rather rapid, while the precise diagnosis of the nature of the neoplasm—sarcoma, carcinoma or lymphosarcoma—cannot be made during life.

*Tuberculous Adenopathies.*—In adults tuberculous adenopathy is of little interest in so far as a differential diagnosis with a mediastinal growth is concerned. Tracheo-bronchial lymph-node tuberculosis, excepting in childhood, never assumes the clinical aspect of malignant mediastinal neoplasms. In children, on the contrary, it may be very difficult to at once differentiate with certainty a large tuberculous tracheo-bronchial adenopathy from a mediastinal lymphosarcoma.

In these circumstances the indications will be furnished by a very thorough examination of the lungs, and by the positive or negative result of the intradermic or cutireaction, as well as by the temperature chart and hereditary antecedents.

*Syphilitic Mediastinitis and Syphiloma.*—A search for recent or old stigmata of syphilis and Wassermann will in most cases settle

the diagnosis. But there are also many circumstances in which a differential diagnosis between a syphilitic adenopathy and a cancerous adenopathy will be impossible to make.

In reality, in both processes the clinical signs may be quite the same. If syphilis be ignored by the patient, if luetic stigmata are entirely wanting and Wassermann negative, no certain diagnosis can be made, so that a differential diagnosis will be out of the question for a certain lapse of time. Specific treatment or the ultimate evolution alone will offer the solution of the problem.

*Abscess of the Mediastinum.*—Cold abscess of the mediastinum alone is of interest from our point of view, as acute abscess of this region offers sufficiently manifest symptoms to be diagnosed with ease.

Certain tuberculous collections of the mediastinum may, on the contrary, present an evolution and symptoms similar to a neoplasm, that is to say, an insidious development, soon acquiring quite a volume and producing marked phenomena of compression.

Should a cold abscess of the mediastinum be presumed to exist other evidences of tuberculosis should be sought for; the localization of the tumor in the anterior or posterior mediastinum should be made out and the data furnished by radioscopy and radiography examined.

*Aneurysm of the Aorta.*—Aneurysms of the thoracic aorta—which may form an intrathoracic tumor of considerable size—present a certain number of symptoms common to tumors of the mediastinum, all indicating phenomena of compression.

These symptoms can be summed up as follows: Pain having variable location—thorax, spine, arms or hands—sometimes similar to the pain of angina pectoris; disturbances of respiration—persistent paroxysmal cough, dyspnœa, etc.; disturbances of phonation—hoarseness, aphonia, etc.; disturbances of deglutition and lastly, dullness and sternal bulging, œdema, cyanosis, collateral venous circulation in the domain of the superior vena cava and unequal pupils.

However, aortic aneurysms generally can be quite distinctly differentiated from mediastinal tumors by other easily recognizable signs, such as a double centre of pulsation, souffles, asynchronism, a delay and inequality between the radial pulses, and lastly, the typical shadow on the radioscopy screen, namely, distinct and often

rounded contours, shadows of thickening due to atheroma of the aortic walls and occasionally pulsation of the aneurysm.

The majority of these signs are naturally wanting in neoplasms of the mediastinum, so that it is clear that the chances of mistake in the diagnosis are relatively small and usually easy to avoid.

*Retro-sternal Goitre.*—The differential diagnosis between this variety of goitre and neoplasms of the mediastinum is principally based upon the fact that in retro-sternal goitre the symptoms of more or less marked compression developing in the respiration, phonation and deglutition, are usually accompanied by mild physical signs, such as limited sternal dullness usually situated very high up, little or no bulging of the sternum, little or no œdema, mild cyanosis and a trifling collateral venous circulation.

On the contrary, these various symptoms are usually quite marked in cases of neoplasm of the mediastinum.

*Mediastinal Pleurisy.*—From its site and anatomical relations a fluid collection developed and enclosed between the two layers of the mediastinal pleura—the so-called mediastinal pleurisy—may be accompanied by the complete mediastinal syndrome—paroxysmal cough, dyspnœa, tracheal tugging, attacks of suffocation, dysphonia, dysphagia, venous stasis and thoracic collateral circulation.

The majority of the physical signs may, on the contrary, be wanting in these cases and it is most uncommon the distinct symptoms are encountered during examination which might aid in making the diagnosis.

If a mediastinal pleurisy is accompanied by acute febrile symptoms or a vomit, the differential diagnosis will naturally be at once settled.

*Dermoid and Hydatid Cysts of the Mediastinum.*—Dermoid cysts of the thorax and base of the neck, developed from the embryonal epithelial elements situated in the median line, usually develop in the middle plane, that is to say, precisely in the mediastinum or its immediate neighborhood.

The physical signs furnished by these growths, always unique and generally having a tardy and very slow evolution, are the cause of much difficulty in differentiating them from malignant neoplasms of the mediastinum.

FIG. 5.



FIG. 6.



FIG. 7.

FIG. 8.





A certain diagnosis of the site and, above all, of the nature can hardly be obtained other than with the X-rays, which alone are capable of revealing the typical image of an intrathoracic dermoid cyst—slightly homogenous shadow, teeth or bone sometimes visible, distinct circular contour and intimate relationship to the median shadow.

Pleural and pulmonary hydatid cysts are less interesting than dermoids in so far as their differential diagnosis with malignant mediastinal neoplasms is concerned. They most usually develop outside of the middle line and mediastinum. They may be multiple, appearing anywhere on the pulmonary pleura and have a relatively rapid evolution.

In these circumstances a radiological examination is alone the means furnishing reliable data as to the site and nature of the cyst. It also may reveal a circular shadow with distinct outlines, accompanied by a dense, completely homogenous opacity.

The radiologic characters of these cysts are clearly very different from those of malignant disease of the mediastinum whose shadow may present most any shape, with ragged edges and varying density as can be seen in Figs. 5, 6, 7 and 8.

#### PROGNOSIS

It is useless to lengthily speak of the prognosis, which is invariably unfavorable. When once the diagnosis of malignant mediastinal neoplasm has been made with certainty, death will not be long delayed.

The evolution of these growths is more or less rapid and death may occasionally ensue relatively early in the process. Thus Case III was extremely short in its evolution. The lapse of time separating the time the first symptoms were noted until death may frequently not exceed a few weeks.

Such instances are, however, exceptional, and a much longer average duration of a malignant mediastinal growth is to be expected, ranging from three to four months at least, up to one year, rarely longer.

#### TREATMENT

No therapeutic measures that have been employed up to the present have given any really favorable results. A large number

of medications have been essayed, that is to say, that none have been satisfactory.

Iodine and arsenic, in particular, as well as their derivatives have been employed in every form and dose, usually without results. Radiotherapy only seems to have given a few temporary successes. In point of fact, the fatally progressive evolution of the process has been known to have been checked momentarily, as for example in Case IV. However, it is to be noted that the X-rays are badly tolerated by certain subjects, as in Case I, in which the thoracic pain, dyspnoea and cough were increased.

Finally, surgical interference has not as yet been successful, excepting tracheotomy as an urgent operation, and this may be most useful in attacks of suffocation resulting from spasm of the glottis.

CASE I.—Female, *æt.* 5½ years, entered the Children's Clinic, service of Professor D'Espine, January 16, 1917.

*Hereditary Antecedents.*—Father well, mother at present in good health. Three other children. The youngest has enlarged cervical lymph-nodes. No still born children.

*Personal Antecedents.*—Born at term, breast fed until eight months of age. First teeth at eleven months. Walked at sixteen months. At the age of two had an abscess of right thigh which was incised. Measles at four and a half years, urticaria at five and a half. Very frequent attacks of bronchitis.

*Present Illness.*—It dates back approximately to September, 1916. Since this date there has been continual cough, especially in the evening and night. The mother thinks that there has been no fever and she has never noticed any enlarged lymph-nodes. No difficulty in respiration or circulation.

The patient was seen for the first time in October, 1916, for bronchitis, and at this time a tumor, probably of the mediastinum, was observed, occupying the anterior mediastinum and upper part of the left hemithorax.

Cutireaction done on October 18 and 20, 1916, was negative both times. Examination of the blood on October 23 gave no particular data, and radioscopy done on the same date showed a very dense shadow throughout the entire mediastinal area and extending one or two fingers' breadth to the right of the sternum (distinct limit),

and to the left up to the mammillary line (irregular outline). (See Fig. 9.)

The entire left upper lobe was likewise obscure. The apex did not become clear either during deep breathing or coughing.

The right lung everywhere perfectly clear, likewise the left lower lobe which, however, was somewhat less transparent than the corresponding right lobe.

The shadow of the heart and large vessels extended two fingers' breadth beyond the right edge of the sternum at the level of the right fourth intercostal space.

The apex of the heart nearly reached the mammillary line in the left sixth intercostal space. The diaphragm was slightly less movable on the left than on the right.

The body weight remained practically stationary from October, 1916, to January, 1917, there being only a loss of 300 grammes during this time.

From the end of October to the end of December, 1916, four radiotherapy séances were given. The first two were well borne by the patient, but with the third disturbances of respiration and circulation arose, *viz.*, distress and dyspnœa, especially in the evening and night, and palpitations with sharp pain in the precardiac area.

The first disturbances did not last and ceased completely when radiotherapy was stopped.

The respiratory distress and thoracic pain on the left were, however, not long in recurring (December 15, 1916), while at the same time several enlarged lymph-nodes made their appearance above the left clavicle and a left subclavicular bulging became apparent.

From this time on the child assumed a distressed expression, the neck became thick, the lower part of the face became notably broadened and the head seemed to sink in between the shoulders.

Likewise from December 15, more cough was noted, as well as effort dyspnœa and cyanosis of the face. The left hand and forearm frequently became violet in color and cold. The child became less and less playful, often asked to go to bed in the daytime and she was often found lying on the bed. The appetite became poor. From time to time rather abundant epistaxis occurred.

*Status Praesens.*—Child of medium height, subcutaneous fat little developed—especially over thorax and upper limbs, musculature limp, skin dry and as if “withered” over the trunk and limbs, mucosa pale, skeleton normal.

Marked contrast between the dryness and bad condition of the nutrition of the trunk and limbs, with the roundness of the face, which was also slightly puffed.

The cheeks full, very red, broad towards the angles of the lower jaw, continued almost directly with upper part of the neck, which was thickened especially on the left by masses of enlarged sterno-mastoid lymph-nodes.

The expression of the face was fixed, the look anxious. Nostrils dilated.

The child, ordinarily very affectionate and good, remained silent, appeared to be exhausted and spoke only one or two words in a low hoarse voice. From time to time, coughing was severe, similar to that of a severe bronchitis. Subfebrile temperature, 99°F.

*Respiratory System.*—The thorax presented a slight bulging in the region of the left subclavicular space and extended, downwards and outwardly, to the level of the third intercostal space in the anterior axillary line.

The left supra-clavicular space was filled by several lymph-nodes the size of large peas. Two of the largest glands, extending up into the sterno-mastoidal area, were not very movable over the subjacent structures, firm in consistency and painless.

In the right supra-clavicular space were two lymph-nodes the size of large peas having about the same consistency as those on the left, but they were quite separate from each other and were very mobile under the skin.

On the anterior aspect of the thorax, near the middle line, was a rather well-developed venous network, especially apparent between the sternum and left breast.

Several very marked veins extended almost horizontally over the posterior aspect of the left hemithorax and then anastomosed a venous network on the posterior aspect of the thorax in the region of the left scapula and scapulo-vertebral sulcus. These veins were notably finer and scarcer behind than in front.

Thoracic amplitude is somewhat greater on the right than on the left. Slight delay in left apex. By palpation no difference could be detected in the pulmonary bases. No exaggeration of the vocal vibrations excepting in the left subclavian space and in the third and second left intercostal spaces close to the sternum where fremitus was greatly increased.

By percussion there was complete dullness in the supra and subclavicular spaces, supra and infra-spinous fossæ and scapulo-vertebral sulcus on the left extending to the spinous apophysis of the fifth dorsal vertebra.

The complete dullness disappeared in front at the level of the fourth intercostal space. Normal pulmonary resonance throughout the left base both in front and behind.

By auscultation, a very strong vesicular murmur which became rapidly attenuated below the nipple and apex of the scapula.

In the left base there was weak broncho-vesicular breathing. Numerous sibilant râles could be heard in both bases.

The respiration was dyspnœic. The dyspnœa increased with the slightest effort. Cough was not very frequent, but deep and very hollow, although there was no expectoration.

The nostrils were largely dilated and did not move with the breathing. The mouth, half opened, sought for air.

*Circulatory System.*—By inspection, very marked pulsations in the epigastric space were seen, but none were visible in the intercostal spaces on the left of the sternum.

By palpation, the pulsations in the epigastric region were very strong, giving the impression of the apex shock. The hand perceived very distinctly the cardiac contractions on the right of the sternum at the level of the fourth and fifth intercostal spaces. Nothing on the left of the sternum.

By percussion, the limits of the heart on the right reached two fingers' breadth beyond the edge of the sternum at the third rib above down to the fifth intercostal below; on the left they were hidden by the sternum. The apex did not extend beyond the left edge of the sternum.

By auscultation an important displacement of the valvular foci to the right was noted. The maximum of intensity of the first sound

was found at the level of the fourth and fifth intercostal spaces along the right edge of the sternum. The beats were forcible, the contractions strong; rhythm regular, no valvular souffles.

Pulse depressible, very regular at 100, equally perceptible on both sides.

Face very red, lips slightly cyanotic. The hands were extremely blue—especially the left—and cold. Very distinct ungual cyanosis.

*Digestive System.*—Tongue clean and moist. Dentition normal. The isthmus of the throat obstructed by enlarged tonsils. Diffuse redness of the pillars, velum and tonsils. The child did not complain of any soreness of the throat, but nevertheless appeared to have some difficulty in swallowing.

Stomach, liver, spleen and intestines normal. Abdomen thin, walls thin. Palpation and percussion negative. Appetite poor. Neither constipation nor diarrhœa.

*Nervous System.*—Pupils equal, reacting well to light and accommodation. No oculomotor disturbances. Patellar reflexes equal, but weak. No Babinski. Motility, sensibility and intellect normal.

*Urogenital System.*—Urine acid, no albumin, sugar or blood.

January 17.—Pulmonary status unchanged. Marked dyspnœa, cyanosis, hoarse cough. The child remained sitting up in bed. Appetite almost *nil*.

January 19.—Dyspnœa considerably increased, likewise cyanosis; cough became very frequent. Persistent orthopnœa. Patient seemed exhausted. Temperature 101°F.

Oxygen inhalations and  $\frac{1}{4}$  c.c. of a 2 per cent. solution of pantopon subcutaneously.

January 20.—Morning temperature 101.5°F. Insomnia. Continual cough. Increasing dyspnœa and distress. Slight dullness was discovered over the right base and fine inspiratory râles extending up to the apex of the scapula.

Cutireaction, done on January 18, remained negative. Fundus of both eyes normal. Urine negative.

January 21.—Temperature ranged from 98.6° to 100.5°F. Souffle in the mid portion of right lung. Fine moist râles throughout the right lung behind and in the extreme left base behind. The

child was rapidly failing, impossibility to sleep or to lie down. Several attacks of suffocation in the night of January 20-21.

Laryngoscopic examination, made extremely difficult from the size of the tonsils, cough and suffocation, did not reveal any paralysis of the vocal cords that might explain the double tone of the voice. The cords were mobile, bright pink and slightly injected. The larynx appeared to be thrown backwards. No deformity or deviation of the trachea was visible to the laryngoscope.

January 22.—Pulmonary status the same as yesterday. Cyanosis notably increased, especially of the face, which was puffed, and the fingers, which were violet and cold.

Pulse small, depressible; radials equal, irregular, 105-115. Cardiac contractions perturbed; heart sound tumultuous. No perceptible valvular souffle. Great exhaustion. Increasingly frequent attacks of suffocation. Death at 9 P.M.

#### AUTOPSY

Cadaver measures 1 metre, 10 centimetres. Weight 15 kilogrammes. Skin very pale. Slight malleolar œdema.

Skull symmetrical, thin, red and transparent. Dura greatly distended. A small non-adherent clot in longitudinal sinus. Cruoric clots in sinus of the base. About 50 c.c. of liquid at the base of the skull.

Brain weighs 1260 grammes. Œdema and considerable injection of the meninges. Small thrombi in left internal carotid, convolutions flattened. Slight increase of the cerebrospinal fluid. Choroid plexuses very dark red. Hypophysis pinkish-gray, the size of a bean. Cerebral substance greatly injected. Central nuclei and cerebellum normal.

*Abdomen.*—Omentum retracted in upper part of abdomen. Transverse colon distended. The liver does not extend below the right costal border. The spleen extends one finger's breadth below the left costal border. On both the right and left the diaphragm reaches the fifth intercostal space. A few cubic centimetres of fluid in the small pelvis.

*Thorax.*—A few cubic centimetres of cloudy fluid in the left pleural cavity. Nothing in the right. The heart is almost com-

pletely pushed over to the right of the median line (see Fig. 10). Slight increase of the pericardiac fluid which contains a few fibrinous shreds.

*Thymic Region* (see Fig. 10).—There exists a large, rounded, nodular tumor which sends off a narrow tongue between the left auricle and ventricle.

On section, this tumor offers an homogenous brilliant, grayish-white surface with small hemorrhages here and there, and areas where the tissue is milky and soft, or else infiltrated by small grayish-yellow granulations the size of a pin's head.

The tumor with its nodules altogether measures eleven by nine centimetres and is enveloped by a smooth, rounded and very tense membrane which penetrates into the upper part of the left pleural cavity where it is very strongly adherent.

The left lung is pushed back by the tumor towards the lower part of the thorax and its apex is adherent to the lower pole of the neoplasm.

The supra-clavicular lymph-nodes on the right form a large bunch; on the left they are smaller and can be felt separately. On section they are grayish and offer a few hemorrhagic spots.

The left axilla contains some very small lymph-nodes without any microscopic change.

The partially compressed large vessels pass through the tumor mass which entirely fills the posterior mediastinum and envelops the aorta and œsophagus to the level of the diaphragm. The tumor does not perforate the vessels at any spot.

The growth strongly adheres to the sternal manubrium, to the internal surface of the ribs and upper part of the dorsal spine.

The organs contained in the neck and thorax are excised *en bloc*.

*Heart*.—Somewhat enlarged. Right ventricle dilated. Tricuspid allows a finger to pass through easily. Pulmonary measures 55 millimetres, myocardium four millimetres thick. Left ventricle contracted and alone forms the apex of the heart. Mitral allows a finger to pass. Aorta measures 44 mm.; myocardium, 12 mm.; firm and light brown in color. All the valves are thin; *foramen ovale* patent.

*Left Lung*.—Pleura rough, covered with a deposit of fibrin and

fibrous thickenings. There are also fibrous formations between the lobes.

Upper lobe pale, containing numerous grayish-yellow foci of broncho-pneumonia.

Lower lobe dark red containing little air. There are also small foci of broncho-pneumonia. The bronchi contain muco-pus. The mucosæ are injected.

*Right Lung*.—Local emphysema. In the lower lobe there is a focus of broncho-pneumonia. Pleura slightly thickened and fibrous. No adhesions.

*Larynx and Trachea*.—On the right side near its bifurcation the trachea is compressed by a nodule of the neoplasm. The lymph-nodes at the level of the bifurcation of the trachea are very large and of the same nature as the neoplastic nodules.

The base of the tongue presents very developed follicles. The tonsils are very large, red and with a smooth surface. The follicles of the pharynx are also very visible.

*Spleen*.—Weight, 60 grammes. Dimensions: 8.5 x 5.5 x 3 centimetres. Surface smooth, slightly tense. On section the splenic tissue is dark red. Consistency quite firm. Follicles clearly visible.

*Left Kidney*.—Dimensions: 9 x 4 x 2 centimetres. Capsule easily stripped off. On section, tissue is light brown, injected, very firm. Pelvis pale.

*Left Suprarenal Gland*.—Thin, poor in lipoid.

*Right Kidney and Suprarenal*.—Same as on left side.

*Liver*.—Dimensions: 18 x 16 x 5 centimetres. Surface smooth. Hepatic parenchyma brownish-red, quite injected, contains one neoplastic nodule the size of a pea. Gall-bladder contains bile; biliary tracts patent.

*Bladder* contracted, mucosa pale.

*Vagina, uterus, ovaries*, negative.

*Rectum*, mucosa pale.

*Colon*.—Contents liquid, mucosa pale.

*Ileum*.—Areas of hyperæmia. Peyer's patches very marked and dark red.

*Jejunum*.—Contents greenish. Hyperæmia localized at the upper part of the folds of the mucosa.

*Stomach.*—Enlarged, containing much alimentary débris. Mucosa pale. Punctiform hemorrhages in fundus.

*Pancreas*, nothing.

*Mesenteric lymph-nodes and lymph-nodes of the hilum of the liver* somewhat enlarged, pink on section.

*Spine.*—In all the vertebral bodies the bone marrow is extensively replaced by a grayish-white tissue, homogenous, transparent, or else grayish-yellow and opaque. The bone trabeculae are everywhere intact. In some very limited areas the bone marrow is in direct contact with the neoplastic nuclei which are adherent to the spine. The upper part of the dorsal spine is slightly convex to the right.

*Anatomical Diagnosis.*—Tumor of the left pleura and anterior and posterior mediastinum. Compression of the large vessels, trachea and œsophagus. Displacement, dilatation and hypertrophy of the heart.

Metastases in the tracheo-bronchial lymph-nodes, in those of the supra-clavicular space on each side, vertebral bodies and liver.

Cedema of the meninges.

Simple enlargement of tonsils. Stasis of abdominal viscera.

Old pleural adhesions of the upper lobes. Slight left fibrinous pleurisy. Broncho-pneumonia.

#### MICROSCOPIC EXAMINATION (PROF. ASKANAZY)

The histologic structure of the tumor is not what might have been expected. In the sections perfectly distinct nerve tissue can be seen, with fibrillary fasciculi interwoven in every direction, exactly corresponding with the nerve bundles.

A certain number of ganglion cells, mostly represented by round or elongated elements with granular protoplasm, are seen in the midst of this tissue composed of nerve fibres with typical nuclei.

The ganglion cells occasionally offer one or two prolongations. Most of their nuclei are eccentric, rarely multiple, and belong to the type of ganglion nuclei. They are round, rather clear and possess a nucleolus.

Some of these cells, very swollen and vacuolate and having lost their shape, are surrounded by a capsule lined by flattened nuclei to the number of six or eight. However, most of them have a single

nucleus. The ganglion cells are always situated in the midst of the nerve fasciculi.

In some parts of this distinctly nervous portion of the neoplasm calcareous incrustations are seen. And lastly, in the midst of the nerve tissue numerous very thin fibrils with small fusiform nuclei are seen, becoming confounded with the nerve fibres in some spots. This is the fundamental tissue of the tumor which extends into the second portion of the neoplasm.

In this second portion, much richer in nerve fibres than the first, one sees a large number of small cells with round nuclei, recalling the cells of the neurologic or lymphoid cells, as well as other larger cells with large nuclei rich in chromatin and usually with little protoplasm.

These very dark nuclei are in certain areas so close together that the cell protoplasm almost entirely disappears, the nuclei only being visible.

The undeniable presence of nerve tissue, as well as the general structure of the tumor, clearly shows that in these areas the neuroblasts are in active proliferation.

In all the other parts of the tumor the cells are extremely small and arranged in groups. A small round-cell sarcoma might be suspected if these areas were not directly mixed with the ambient neoplastic tissue. The purely nervous parts and those where the neuroblasts exist are, in fact, formed by foci penetrating each other.

The ganglion cells, stained with Unna-Pappenheim, can be distinguished by the bright red color of their protoplasm from the small cells which only show a narrow zone of pink protoplasm around a dark blue nucleus.

Stained with Giemsa, the nerve parts of the tumor are stained light yellow and a very few cells are undergoing mitosis (dyaster).

*Thymic Region.*—Some bits of tumor removed from the thymic region contain many small cells of lymphoid aspect, varying in size and enclosed in a thin stroma through which fibrous trabeculæ and numerous blood-vessels pass.

Near the centre of the neoplasm several foci of necrosis are visible. At no spot could any débris of the thymus be discovered.

*Pathological Diagnosis.*—Malignant ganglioneuroblastoma.

*Summary.*—In this case, the patient, aged five and a half years, presented symptoms of an affection of the mediastinum about five months before death.

After a latent phase of indeterminate length, the first symptoms—in appearance very mild—appeared in the respiratory tract—phenomena of slight tracheo-bronchial irritation.

About three months elapsed between the first symptoms and the development of the serious disturbances arising in the respiratory and circulatory systems—phenomena of compression strictly speaking.

The symptoms revealing the presence of a mediastinal growth and continuing with its evolution were, in this case, those that have been noted in neoplasms of the mediastinum, namely, cough, progressively increasing dyspnœa, attacks of suffocation, cyanosis, œdema of the upper part of the body and a thoracic and abdominal collateral venous circulation. Retro-sternal pain, often noted, did not occur in this case.

The evolution of the tumor was rapid. Death, hastened by broncho-pneumonia, resulted from strangulation.

Autopsy showed the presence of a relatively large, rounded, nodular tumor of the mediastinum, homogenous and grayish-white on section, starting in the pleura, pushing aside the left lung and heart and partially compressing the large vessels, trachea and œsophagus.

Microscopic examination of various parts of the neoplasm revealed the unexpected presence of nerve tissue with typical fibres, ganglion cells and cells recalling those of the neuroglia and lymphoid cells. Neuroblasts were found undergoing proliferation and the pathologic diagnosis was malignant ganglioneuroblastoma. No element of the thymus gland could be discovered anywhere in the tumor.

CASE II.—Male, *æt.* 12 years, was first seen on June 6, 1905, for enlarged lymph-nodes in the neck. No other subjective phenomenon was at this time present. The patient had been treated for a certain time for a lesion of the upper left lobe, characterized by considerable dullness, souffle and bronchophonia. As no Koch's bacilli were found, the diagnosis of simple pulmonary sclerosis, chronic pneumonia with dilatation of the bronchi and enlarged lymph-nodes of the neck had been made.

In October, 1905, after arsenical treatment the child was sent to

Cannes. On March 16, 1906, Doctor Revillet, of Cannes, sent the following report to Professor D'Espine:

"Left apex, which was dull in front and behind, was so suspicious upon the arrival of the patient that sea bathing was not allowed.

"About January 20, 1906, the temperature, which had ceased, reappeared, with nocturnal sweating, oppression, pain in the left shoulder and pleural pain below the nipple. Cough very frequent. Depression and progressive weakness. Increase of the auscultation and percussion signs. Temperature ranging from 99°F. in morning to 102.2°F. in evening."

Early in February, 1906, Doctor Revillet noted that the entire upper third of the left lung was dull, with bronchial souffles, and fine subcrepitant râles. General condition very bad. Oppression. Cyanosis of face. Evening temperature ranged from 100.6°F. to 101.2°F. Nocturnal sweating, anorexia.

The diagnosis at this time was tuberculous infiltration of left apex, caseous pneumonia undergoing softening and bronchial adenopathy on the right.

The child was then brought back to Geneva, and on March 13, 1906, Doctor Dutrembley sent the following report to Professor D'Espine: "The child is cyanosed and very dyspnoic, cannot lie down, remains seated with the legs hanging down, the elbows on a pillow and the body bent forward.

"On both sides of the neck one can feel a chain of lymph-nodes extending to the base of the neck where they dip into the thorax. Some are as large as walnuts.

"Percussion of the thorax gives dullness over both apices, especially behind as far as in the infra-spinous fossæ.

"Auscultation reveals a souffle, more marked behind than in front, in both upper lobes. No râles can be detected even when the patient coughs. Some râles and pleuritic friction-sounds in the middle and lower portions of the right lung.

"Cough is dry and rather frequent. Very profuse mucous expectoration. Pulse small, rapid. Temperature 100°F. in the evening. Since March 11, œdema of the lower limbs."

Injections of sodium cacodylate appeared at first to slightly relieve the dyspnoea.

During May a consultation was held and a diagnosis of malignant tumor of the mediastinum was made.

From this time on the orthopnœa continued and the child could not lie down. Finally, the abdomen became distended and death ensued on May 31. Autopsy, by Professor Askanazy, was done the following day.

*Autopsy* (see Figs. 9 and 10).—The thoracic organs were removed *en bloc*.

A very large tumor situated in the anterior mediastinum descends to the apex of the heart, pushes aside both lungs and extends backwards to the trachea and bronchi.

The posterior mediastinum is intact in the upper half of the thorax while the neoplasm approaches the œsophagus in the lower half.

The growth recalls the shape of a very enlarged thymus or that of a triangle with blunt angles. Its base is in the mid-line below, while its sides rise up to the neck, pushing aside both pulmonary apices and at the same time depressing them.

The base of the tumor—the lower horizontal part—measures 15 centimetres, the sides 17 to 18 centimetres; the antero-posterior diameter measures 12 to 13 centimetres.

The lateral aspects of the growth are covered by the mediastinal pleura and are slightly nodular. A large number of nodules project above the surface of the tumor and many are isolated from each other. Some nodules also exist above the diaphragm.

By displacing the heart to the right two nodules are seen on the internal surface of the pericardium and surround the pulmonary veins. A small nodule, the size of a lentil, lies close to the aorta, while another is seated on the parietal pericardium opposite the pulmonary artery. A neoplastic patch projects between the aorta and inferior vena cava.

The neoplasm slightly pushes the trachea to the right. Some hemorrhages are visible on the parietal pericardium. The right lung presents fibrous adhesions and a few small hemorrhages.

Some adhesions are found on the left lung, which at the level of the hilum is a tumor the size of a walnut. A neoplastic nodule the size of a pea composed of whitish tissue is found in the paren-

FIG. 9.

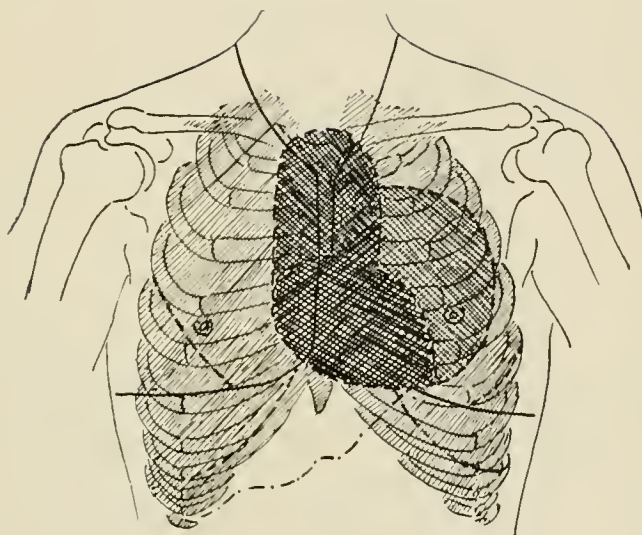


FIG. 10.

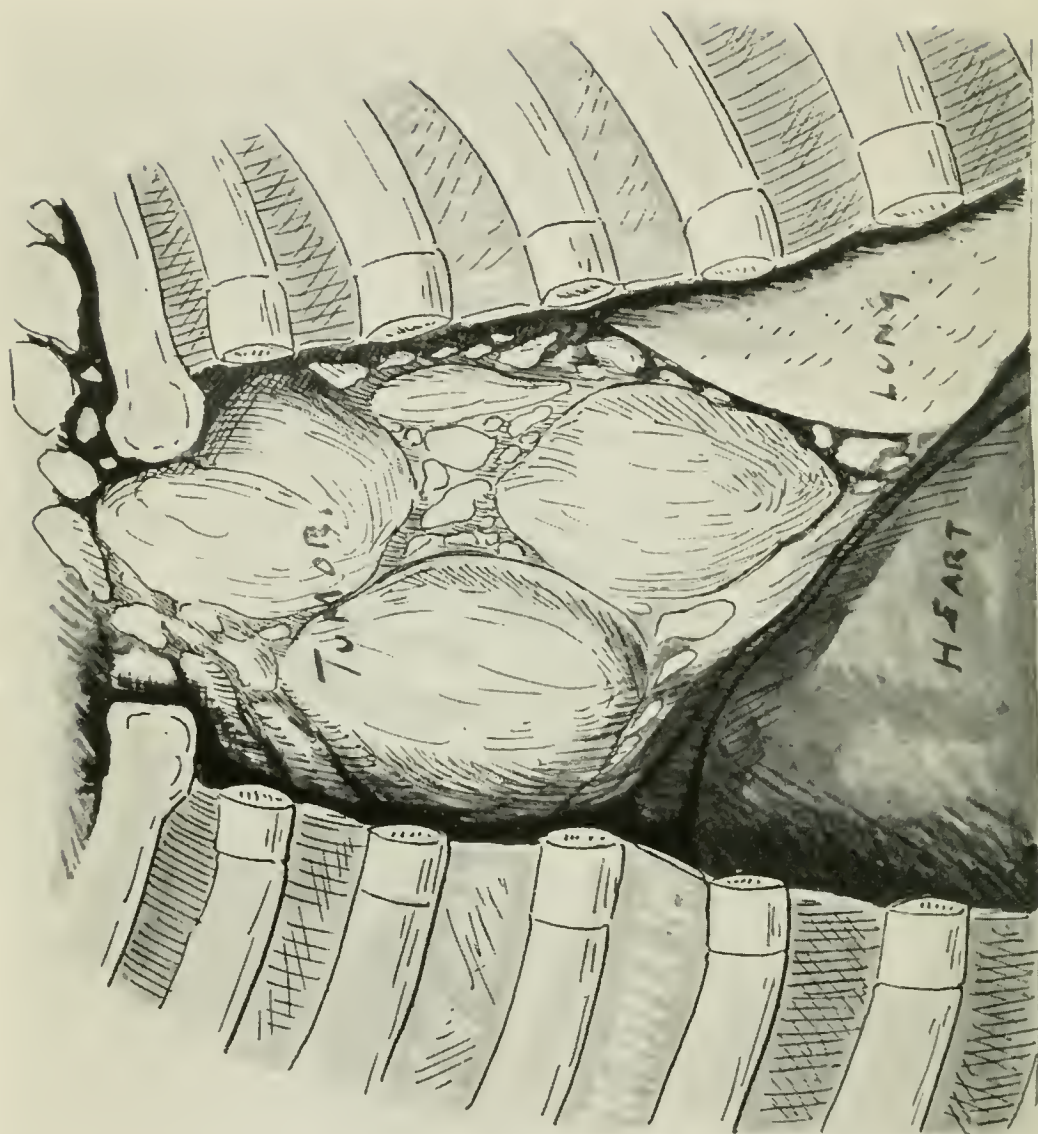
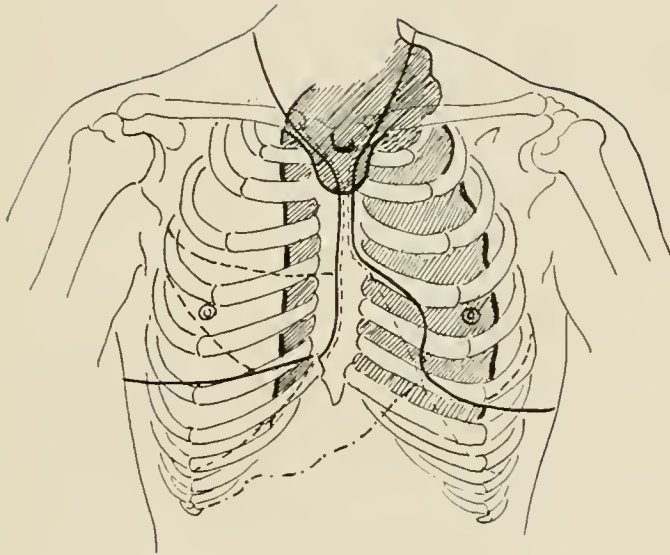


FIG. 11.





chyma of the lower lobe of the right lung. On section, the tissue composing the neoplasm is pinkish white and quite compact.

*Microscopical Examination.*—A number of bits taken from various parts of the neoplasm were examined. The histologic structure of the tumor is relatively uniform throughout.

It is lymphatic tissue composed of small lymphoid cells, intermixed with very vascular fibrous areas. The lymphatic parenchyma itself is in places the site of fibrous trabeculæ forming a sort of network whose meshes are filled with lymphocytes. The neoformed connective tissue assumes a hyaline character in some spots. One also sees some large cells, irregularly distributed in the midst of the tissue.

These large cells are rich in protoplasm, contain one or several nuclei darkly stained, and resemble elements met with in lymphogranulomata. Although their aspect also recalls that of giant cells, no evidence of tuberculosis can anywhere be detected.

No Hassal's corpuscles can be found, but this fact cannot completely exclude the possibility of a thymic origin of the neoplasm. The principal tumor has exactly the shape of the thymus. The hypothesis of a lymphogranuloma can also be discarded. There are no visible eosinophiles or cells with a large nucleus (Sternberg).

*Pathologic Diagnosis.*—Lymphosarcoma of the anterior mediastinum, apparently of thymic origin. Thoracic and pulmonary lymph-nodes with metastases.

*Summary.*—In this twelve-year-old patient the first symptoms also developed in the respiratory tract about one year before death.

As in Case I, all the symptoms at the onset were relatively mild and even misled the diagnosis until a short time before death.

It was only about two months before death that more typical clinical symptoms led one to suspect a malignant tumor of the mediastinum, viz., cyanosis, dyspnœa, orthopnœa, thoracic pain and enlarged supra-clavicular and cervical lymph-nodes.

The absence of characteristic symptoms in some instances of tumor of the mediastinum is remarkable as in this case in which the mediastinal tumor and compression of the thoracic organs were considerable.

At autopsy a very large rounded, firm tumor with a nodular

surface was found, seated in the anterior mediastinum, completely pushing both lungs backwards, compressing the trachea and covering the pericardium.

Microscopical examination demonstrated the existence of tissue of lymphatic nature with numerous small lymphoid cells and large neoplastic cells. No eosinophiles or Sternberg's large nucleus cells.

The diagnosis of lymphosarcoma of possible thymic origin was made, even with the absence of typical cells of the thymus gland—Hassal's corpuscles, cortical or medullary cells—because of the shape and site of the neoplasm.

CASE III.—Male, *æt.* 16 years, entered Surgical Clinic on March 3, 1916. Hereditary and personal antecedents devoid of interest.

The onset of the present illness began on February 26, 1916, when, in the morning while dressing, the patient noted that his neck was swollen. Two days later (February 28) a physician was consulted because the neck was still larger. He was ordered an ointment and arsenic internally. Cold caused headache.

Cough began on March 1st, the voice became hoarse, there was difficulty in breathing and cold caused headache.

*On admission* to hospital the patient looked apparently well with well-developed muscles. Some cyanosis, dyspnœa, hoarseness; pulse, 120; temperature, 98.6°F.

On the right side of the neck in front of the sternomastoid a tumor the size of a tangerine orange, hard, uneven, strongly adherent to the subjacent structures, painless and non-pulsatile, was found. A few movable small lymph-nodes were found beside the tumor.

The skin of the affected region was violet color with a very marked varicose ectasis of the superficial veins. The thorax itself and the upper part of the abdomen as far as the level of the ribs presented small venous dilatations irregularly distributed.

In the right axilla were some enlarged, hard, adherent lymph-nodes, but painless. In the groins some disseminated lymph-nodes. Nothing could be detected in the left axilla.

*Nervous System.*—Pupils equal, with normal reflexes. Patellar reflex absent on right, slight on the left. No Babinski, no clonus. Sensibility and motility normal.

*Circulatory System.*—Heart beats regular, heart normal in size.

No souffle or superadded bruits. Auscultation hindered by respiratory bruit. Radial pulse equal on both sides. High blood-pressure.

*Abdomen.*—Stomach negative. Spleen can be percussed. Liver one finger's breadth below costal margin. Intestine and stools negative.

*Urogenital System.*—Urine normal, acid reaction. Albumin, sugar, indican absent.

*Respiratory System.*—Dullness over both apices with blowing respiration. Disseminated ronchi.

*Blood:*

Hemoglobin (Sahli) .....	75 per cent.
Red cells .....	4,030,000
White cells .....	9,450

Coagulation begins in 9½ minutes, ends in 12 minutes.

March 6.—No improvement. Quite frequent attacks of suffocation. A marked dullness found over the entire sternal region.

*Radiography.*—See Fig. 5.

*Biopsy.*—One lymph-node removed, was found pinkish-white on section, with microscopically a normal structure. Great dilatation of the lymphatic vessels at the hilum of the lymph-nodes. Intraglandular lymphatic sinus somewhat enlarged with endothelial cells and lymphocytes. Some red blood corpuscles. No neoplastic tissue.

*Pathologic Diagnosis.*—Lymph-node with lymphatic stasis of the vessels of the hilum and catarrh of the sinuses.

March 8.—*Laryngological Examination.*—Mucosa of pharynx somewhat red. Vocal cords congested, hardly tumefied, movable during phonation and deep inspiration.

Arytenoids distinct and movable. Nothing to note on the posterior and lateral walls.

A slight projection is seen in the cricoid area which below appears to extend into the trachea. It is impossible to follow it further.

During quiet breathing—the vocal cords being largely separated—there is inspiratory effort.

The neck is considerably swollen down to the suprasternal fossa. Venous circulation hindered. Compression of the trachea.

March 9.—At 7 P.M. severe attack of suffocation requiring immediate tracheotomy, after which patient breathed relatively better.

March 10.—Death at 1:40 A.M. from an attack of suffocation.

*Autopsy.*—Young male, with good musculature. Very distinct thoracic and abdominal subcutaneous venous network. Neck hard, tumefied, no œdema. No enlarged lymph-nodes.

Skull smooth and symmetrical. Sinuses filled with liquid blood, dura congested, ventricles not dilated, substance of brain and cerebellum firm.

*Abdomen.*—Intestines distended, appendix normal, venous network not apparent. Pecquet's cistern tumefied containing an opalescent fluid.

*Thorax.*—The right pleural cavity contains about 100 c.c. of brownish fluid. Nothing in the left pleura.

A large tumor, intimately adherent to the posterior aspect of the sternum prevents the hand from being passed from one lung to the other.

It is necessary to saw the clavicles in order to disengage the visceral mass at the base of the neck. After this the organs of the neck and thorax were removed together.

A neoplastic lump, the size of a large apple, adherent on the right side to the principal thoracic tumor itself, forming a single mass between the right heart—which it partially covers—and both lungs, was found.

Dissection of the internal jugulars and superior vena cava showed that the tumor compressed these vessels, flattening them.

The tumor even penetrated the lumen of the right internal jugular and superior vena cava, forming smooth, pink, slightly projecting patches on the internal aspect of these vessels. No adherent thrombus. A search for the thoracic duct remained fruitless.

*Lungs.*—On section, simple stasis. The right pleura was strongly adherent at its antero-internal border. A few subpleural nodules. Also a few others in the lymph-nodes of the hilum. Nothing on the left.

*Heart.*—Myocardium normal color. Right ventricle, 0.6 centimetre; left ventricle, 0.4 centimetre. Aorta, 5.2 centimetres.

*Stomach* distended. A few hemorrhagic spots in the mucosa.

*Intestine.*—Mucosa normal. No lesion.

*Spleen.*—Rather movable. Hypertrophy of follicles.

*Kidneys.*—Numerous subcortical metastases, the size of a lentil to a pea. Hypertrophy of cortex.

*Suprarenals.*—Nothing.

*Liver.*—Pale, slightly yellow in color, traces of fatty degeneration. Gall-bladder negative.

*Urogenital system,* negative.

Marked hypertrophy of the papillæ at the base of the tongue. Hypertrophy of tonsils. No varices of œsophagus. Thyroid distinctly contained in its fibrous capsule.

Section of tumor: Fundamental color gray-pink, with yellow areas of necrosed tissue in some spots.

*Pathologic Diagnosis* (see Fig. 11).—Sarcoma of mediastinum having invaded the base of the neck, especially on the right. Perforation of the superior vena cava and right internal jugular. Right pleural and pulmonary metastases. Renal metastases.

Status thymico-lymphaticus, tumefaction of follicles of the spleen and base of the tongue and tonsils. Slight fatty degeneration of the liver.

Hyperæmia of the meninges. Dilatation of the sinuses. Hypoplasia of the aorta. Sclerosis of the presternal subcutaneous tissue.

*Microscopic Examination* (Prof. Askanazy).—One can see veritable areas of small lymphoid cells with little protoplasm and relatively very large nuclei. These cells offer the type of small lymphocytes and are pressed together in the meshes of a slightly developed stroma.

Around the vessels there is either a thin reticulum or merely fine tracts of connective tissue.

Certain areas appear to be completely necrosed and here the cells and nuclei can only be vaguely made out, the latter being almost completely effaced.

There is no inflammatory reaction at the limits of these foci of necrosis.

No recognizable thymic elements. No eosinophiles. No cells with large nuclei (Sternberg).

*Diagnosis.*—Lymphosarcoma. The diagnosis of lymphogranuloma can be unhesitatingly discarded.

*Summary.*—Here is the case of a sixteen-year-old boy who himself noted the first symptoms of the process only twelve days before death.

The tumor had evidently gone through a latent phase whose total duration is unknown.

The first sign that attracted the patient was an increase in the size of his neck (lymph-node? venous stasis?).

Immediately after ordinary phenomena of compression of the upper respiratory tract, large vessels and nerves ensued—cough, dyspnoea, cyanosis, hoarseness, and attacks of suffocation.

The diagnosis of mediastinal tumor was readily made from the symptoms offered, the considerable sternal dullness and lastly, the extremely distinct X-ray picture (see Fig. 5).

Autopsy revealed a rather large tumor seated in the anterior mediastinum, covering the pericardium, compressing and penetrating the right internal jugular and superior vena cava.

Microscopically, the growth was made up of small, very numerous lymphoid cells contained in the meshes of connective tissue. There were no thymic cells or eosinophiles. The diagnosis was clearly that of lymphosarcoma of the anterior mediastinum.

CASE IV (see Figs. 6 and 7).—Female, *æt.* 27 years, entered the Surgical Clinic of the University, August 25, 1915. Hereditary and personal antecedents negative, other than the patient's mother died at the age of sixty-eight from cancer of the rectum.

*Present Illness.*—In February, 1915, had influenza, which kept her in bed a fortnight. She then returned to her duties as a teacher, but nevertheless from this time she occasionally had a pain in the left supra-clavicular region.

Suddenly in July, 1915, the patient was compelled to give up her work on account of violent pain in the left cervical region with very sharp irradiations in the left arm and back. At the same time there was severe headache on the left side of the head which lasted two days, and from this time on the patient could hardly turn the head to the left.

A tumor, extending upward along the sternomastoid muscle appeared at the same time as the painful phenomena. It was the size of a hen's egg, hard and not very painful and soon reached the sternal region where it projected, while a large venous network became apparent over the entire chest and left shoulder, down to the elbow.

Shortly after this, the patient was seized by vomiting immedi-

ately after meals. She had the sensation as if she had swallowed something too large.

*Examination* (August 25, 1915).—A somewhat thin woman with a slightly bronzed tint.

By palpation a bulging in the sternal region was detected. The tumefaction was warm, painful and dull on percussion. In the left cervical region a painless, deeply adherent tumor the size of a hen's egg could be felt. There were some palpable lymph-nodes in the left axilla. Nothing on the right side.

The sternal tumor was superficial, soft, painful and seemed to continue down into the mediastinal region and upwards as far as the supra-clavicular fossa. A very developed thoracic venous network was especially evident on the left side of the chest.

The patient complained of painful irradiations throughout the sternal region, as well as in the left clavicular and cervical regions and in both arms down to the hands.

*Heart* did not appear to be displaced. A very localized inorganic mesosystolic murmur could be heard.

*Lungs*.—The respiration was completely absent in the left lung from the apex to about the third rib in front and to the spine of the scapula behind. On the contrary, the respiration was increased at the base. No râles, no crackling. Right lung negative.

*Digestive System*.—Difficulty in swallowing solid food. There had been some vomiting, but at present there was no particular digestive disturbance. Liver and spleen normal. Some constipation, never any diarrhœa.

Patient stated that she had lost sixteen pounds in one month, but this was due to vomiting and difficulty in taking solid food.

*Urogenital System*.—Menstruated at fourteen normally. Urine acid, no albumin, sugar or indican.

August 25.—Blood Examination:

Red cells .....	4,650,000
White cells .....	13,387
Polynuclears .....	75 per cent.
Lymphocysts .....	22 per cent.
Large mononuclears .....	3 per cent.
Wassermann .....	Negative
Hemoglobin .....	90 per cent.

*Radioscopy*.—A tumor can be seen occupying the left supra-

clavicular fossa, the left half of the thorax, the mediastinum and extending two fingers' breadth beyond the right edge of the sternum (see Fig. 11).

The tumor is quite distinctly limited in the neighborhood of the right edge of the sternum, likewise to the left apex. Elsewhere it becomes confounded with the adjacent organs.

August 26.—Patient cannot sleep on account of pain in the shoulders irradiating down the arms. Ice-bag on sternum.

August 27.—Ice-bag could not be tolerated by patient. A little blood expectorated.

August 28.—A new tumor has appeared in the right cervical region. It is hard, deeply fixed, hardly movable and painful.

August 29.—A small fluid collection detected at base of left lung, with a souffle at inspiration and expiration. Aphonous pectoriloquy. Compression souffle in apex of same lung. No râles, no pain.

August 30.—Blood examination:

Red cells .....	5,560,000
White cells .....	14,700
Polynuclears .....	75 per cent.
Lymphocysts .....	23 per cent.
Mononuclears .....	2 per cent.
Hemoglobin .....	90 per cent.

An exploratory puncture at the left base was also made and about 10 c.c. of yellow, slightly cloudy liquid was withdrawn containing fibrinous shreds.

Chemically, this fluid was alkaline, containing much albumin, but no blood. The cytological examination gave the following result: Lymphocysts, 75 per cent; polynuclears, 25 per cent. A few epithelial cells, no bacteria, no neoplastic cells. After centrifugalization, some of the deposit was injected into a guinea-pig.

August 31.—The patient was relieved by the pleural puncture, cough ceased, likewise the pain.

The séances of radiotherapy, which had been begun on August 27, appear to have acted favorably. The rays were given for twenty minutes at a distance of fifteen centimetres, once daily, the hard, filtrated rays being used. On one day the left clavicular region, the next day the sternal region, etc.

The patient was seen by Professor Girard, who suspected a lymphosarcoma of mediastinal origin or a metastasis from a

hypernephroma. On the other hand, although the Wassermann was negative, KI and inunctions with a 4 per cent. Hg. oxychloride ointment were ordered.

September 1.—The tumor located in the sternal region appeared to be still adherent to it and was very painful when palpated. The skin covering it was still hot and normal, but a very developed venous network existed in it.

September 3.—Condition stationary. Inunctions with white precipitate ordered.

September 5.—X-ray treatment continued. As the pulse remained rapid, five drops of digalene were ordered daily.

September 6.—Sodium salicylate 50 centigrams twice daily. Condition stationary.

September 7.—Radiotherapy stopped.

September 9.—Since X-ray treatment has been stopped the patient has suffered considerably. No change in local condition; the anterior dullness remains the same, while the resistance above the left clavicle has slightly increased.

September 12.—As the patient suffered still more, the X-rays were again resorted to. The pain irradiates to the elbow, sometimes even to the fingers on the right.

September 13.—Pain on the left of the sternum. Palpation very painful at this point, while up to this date the maximum site of pain had been on the right of the sternum.

September 14.—Pain above the right clavicle, where a painful mass the size of a walnut, with rather diffuse limits, can be felt. Fowler's solution ordered.

September 16.—Slight improvement since the X-rays have again been given.

September 18.—There is less resistance above the right clavicle. Condition on left side unchanged.

September 22.—Blood examination:

Red cells .....	3,263,000
White cells .....	7,207
Lymphocysts .....	23 per cent.
Large mononuclears .....	3 per cent.
Small mononuclears .....	1 per cent.
Transitional .....	10 per cent.
Hemoglobin .....	85 per cent.

September 29.—Heart slightly displaced to right. At the apex the limits are more distinctly heard over the xiphoid appendix than in the fifth left space.

Percussion of left lung reveals dullness extending backward to underneath the spine of the scapula. Respiration whistling, while the vesicular murmur is absent throughout the supra and infra-spinous fossæ.

In front the dullness extends to just under the clavicle. On the right it reaches three fingers' breadth from the middle line.

Slight œdema of left hand and forearm. Spasm of œsophagus during deglutition so that only liquids can be taken.

October 2.—Pleural puncture withdrawing 650 c.c. of yellowish cloudy fluid.

October 5.—Second puncture withdrawing 1350 c.c. of reddish cloudy fluid. Five c.c. of this fluid were injected subcutaneously in the patient.

October 8.—X-rays again stopped because they distressed the patient. On the right, above the sternal fourchette and under the sternomastoid a hard tumefaction, the size of a walnut, painful on pressure, can be felt.

October 9.—Third puncture withdrawing 1200 c.c. of fluid.

October 14.—X-rays recommenced.

October 15.—Increase in the size of the small, hard painful lymph-node detected a fortnight previously in the right supra-clavicular region.

October 20.—A second lymph-node detected about 2 centimetres distant from the precedent node.

October 28.—Biopsy of a lymph-node. Small homogenous tumor the size of a pea, white, with a slight central vascular network. Microscopically, layers of round cells of medium size with numerous mitoses. Capillary network strongly developed. Diagnosis: *Round cell sarcoma*.

November 8.—X-rays recommenced.

November 22.—Patient becoming exhausted.

December 3.—Profuse sweating. Ptosis of upper left lid, pupils unequal. Atropine, ordered for sweating, stopped. The ocular phenomena then subsided.

Death on December 23.

*Autopsy.*—Rather thin female. No œdema or rigidity. Neck very large; on right an old cicatrix. Skin of upper half of thorax pigmented with one or two stellate cicatrices above the left breast. A small tumor, the size of a chestnut, projects under the skin over the mediastinal region.

*Cranial Cavity.*—Hyperæmia of all the organs. Nothing else to mention.

*Abdomen.*—Slight intestinal distension. Duodenum dilated. Liver extends one finger's breadth below costal margin. Diaphragm reaches the level of fourth intercostal space on the right, to the level of the fifth rib on the left. Two hundred c.c. of clear reddish fluid in true pelvis.

*Thorax.*—At the level of its lower half the sternum is transpierced by a homogenous, soft tumor, similar to lymph-node tissue and this projects under the skin. In its lower third the sternum is infiltrated and friable, sclerosed in its upper third.

The anterior mediastinum is entirely filled by a dense neoplastic mass, adherent to the sternum and to the first ribs on the left. The ribs are partially infiltrated and break easily. No thymus can be found.

The neoplasm extends up on both sides of the neck into the supra-clavicular fossæ in which numerous lymph-nodes are found.

Nothing particular to note in the organs of the neck excepting an invasion of the wall of the trachea by the neoplasm over a small extent.

The left pleural cavity contains about 700 c.c. of clear, reddish fluid.

The left pulmonary apex is totally invaded by the neoplasm and adherent to the parietal pleura. The remainder of the organ is the seat of splenization due to a diffuse neoplastic infiltration and the pulmonary parenchyma is completely deprived of air.

The right lung, which is almost intact, there merely being a few subpleural metastases, is the seat of œdema and hyperæmia.

The bronchi are compressed by the growth. The lymph-nodes of the hilum are respected by the neoplasm which, on the contrary, invades and perforates the left pulmonary veins.

The parietal pericardium is completely invaded by the tumor.

The visceral pericardium presents very numerous metastases and the heart is villous. Little pericardial fluid.

The heart, with numerous subpericardial metastases, is soft. Left ventricle: 8 to 10 cm. Endocardium and valves offer nothing abnormal.

*Vessels of the Base of the Neck.*—The left brachiocephalic trunk is completely occluded by the neoplasm. Its lumen cannot be distinguished.

The arch of the aorta is somewhat narrowed.

The superior vena cava, right brachiocephalic trunks, both arterial and venous, are intact, likewise the left carotid and subclavian.

The left sympathetic is, on the other hand, comprised in the growth which is adherent to the last cervical and first dorsal vertebræ.

*Spleen.*—Soft, measures 12 x 5½ x 2 centimetres.

*Kidneys.*—Quite numerous metastases in cortex.

*Suprarenals.*—Normal.

*Liver.*—Nutmeg from stasis.

*Pancreas.*—Normal.

*Intestine.*—Gastrointestinal catarrh.

*Left Ovary.*—Metastasis the size and shape of a billiard-ball. Small metastasis in Douglas' pouch.

*Genitalia and rectum* normal.

*Diagnosis.*—Large polymorphous cells sarcoma of anterior mediastinum, probably originating in the thymus.

Invasion of sternum, first left ribs, pleuræ, pericardium, trachea, left pulmonary veins and sympathetic.

Occlusion of left brachiocephalic venous trunk, narrowing of aorta, heart soft, nutmeg liver from stasis, gastrointestinal catarrh.

Pulmonary, subpericardial lymph-node, subclavian, renal and ovarian metastases, with one in Douglas' pouch.

#### MICROSCOPICAL EXAMINATION (PROF. ASKANAZY)

In the midst of a rather weakly developed fibrous stroma can be seen large masses of cells, some small like lymphocytes, others a little larger.

The largest cells present a very distinct protoplasm with a clear

nucleus. These large cells with a large nucleus rich in chromatin, are nevertheless exceptional.

Besides, numerous vessels with thin walls exist in the midst of the neoplastic tissue. One can even see a vein occluded by a neoplastic thrombus. The thrombus is composed of fibrous tissue, a mass of small lymphoid cells and a few polynuclears. Numerous small necroses can be recognized in the midst of the thrombus.

The proliferation of the neoplastic tissue in the interior of the vein can be followed in several serial sections and at one point may be seen the direct penetration of the tumor elements into the lumen of the vessel.

Finally, at some spots the connective tissue stroma becomes distinctly hyaline.

At every point where the tumor penetrates the pulmonary tissue the anthracotic pigmentation of this tissue can be readily recognized and the passage of the neoplastic cells into the lumen of the alveoli can be seen. In fact, there exist certain zones in which the alveolar wall is more or less thickened and where the lumen of the alveoli is filled with a tissue freely developing within them.

Lymphoid elements are everywhere to be found.

In some parts the neoplastic cells are all rather large in size and present themselves in the form of polymorphous elements. Nevertheless, the large cells are very rare.

No visible thymic elements. No eosinophiles, no cells with large nuclei (Sternberg) which might characterize the neoplasm as a lymphogranuloma.

*Diagnosis.*—Lymphosarcoma.

*Summary.*—In this case we are dealing with a female of twenty-seven years. The affection in the mediastinum became manifest five to six months before death, following an attack of influenza, by pain in the left cervical region and arm and back, dysphagia, the development of cervical lymph-nodes and a venous network on the thorax.

The diagnosis of mediastinal tumor could be easily made from the symptoms—which were sufficiently typical—and radiography.

Besides, a biopsy of a supra-clavicular lymph-node made two months before death allowed one to make the exact diagnosis of the nature of the growth.

In this case there was but little dyspnœa and the phenomena of compression of the respiratory tract were relatively slight. On the other hand, dysphagia appeared early in the process, as well as very marked painful thoracic phenomena.

Autopsy revealed a medium-sized tumor in the anterior mediastinum, perforating the sternum and invading the parietal and visceral pericardium, left lung, the pleuræ, trachea and left sympathetic.

Microscopically, lymphoid cells were found contained in a thin stroma with a few rare neoplastic cells of large size. In no part of the tumor could any elements of the thymus or eosinophiles be detected. The diagnosis was lymphosarcoma.

CASE V.—Male, *æt.* 27 years, entered hospital on March 9, 1914. Father died at the age of forty-eight from a sarcoma.

The patient has had a deformity of the thorax since childhood. Syphilis not admitted. Usually in good health.

*Present Illness.*—Patient “took cold” in December, 1912, since which time he has coughed continually. During the summer of 1913 there was some improvement, but the cough returned in the autumn and was accompanied by dyspnœa. A physician was consulted in November, 1913, who only found a bronchitis, but suspected a mediastinitis or tumor of the mediastinum. In fact, at this time the veins on the thorax were greatly dilated and there was percussion dullness over the sternum.

In December, 1913, and January, 1914, cough was severe, likewise the dyspnœa. At the beginning of February, the cough, and especially the dyspnœa, increased and the patient was seen by Professor Bard, who diagnosed a malignant growth of the mediastinum, and ordered radiotherapy.

On February 7, another physician was consulted who diagnosed a syphilitic (Wassermann +) mediastinitis, but as the patient absolutely denied acquired syphilis, a diagnosis of congenital syphilis was made.

The exhibition of KI and radiotherapy were followed by a very slight improvement.

Nevertheless the dyspnœa progressively increased and the patient decided to enter hospital with the diagnosis of mediastinitis.

*General Status.*—Man of small build, scoliosis with convexity

to the right and deformed thorax—a projecting angle formed by the ribs of the right hemithorax—and arched sternum. Subcutaneous veins of thorax enormously dilated.

In the right submaxillary region and extending down to the clavicle, was a large, very painful mass, but without fluctuation. The skin covering it was normal.

Patient pale, slightly cyanosed, very dyspnoëic and coughs considerably. Expectoration purely mucous in nature. Subject is very thin, musculature flabby, and a little œdema of the face. Subcutaneous veins of abdomen very developed.

*Respiratory System.*—Considerable deformity of thorax behind. Dorsal scoliosis, convexity to the right, with vaulting of the right hemithorax. In front, the sternal region corresponding to the mediastinum is strongly projecting, especially on the right side where a tumefaction in the region of the neck likewise exists. Subcutaneous veins extremely dilated.

Percussion reveals dullness over a great extent on both sides of the middle line.

Auscultation—difficult on account of the thoracic deformity—reveals a tubal souffle in both apices and lower down. No râles. Strong vibrations.

Behind, in the middle line, strong inspiratory and expiratory souffle extending beyond the spine on each side, especially on the right. Resonance preserved throughout on both sides. Respiration rough, but no souffles or râles. D'Espine's sign absent.

Severe, continual cough. Abundant mucous expectoration.

*Circulatory System.*—Peripheral arteries soft. Pulse weak, can hardly be felt, 120. The apex of the heart is in the sixth intercostal space outside of the mammillary line. Rhythm regular. Sounds very dull. No appreciable murmur.

*Nervous System.*—Pupils large, left more dilated than right. Reacting poorly to light and accommodation. Tendon reflexes very weak on each side, especially the left. No clonus, no Babinski.

*Urogenital System.*—Gonorrhœa three years previously, no complications. Syphilis denied. Urine cloudy, containing urates. No albumin, sugar or blood.

*Digestive System.*—Anorexia. Tongue dry, red, parched. Ab-

domen soft, subcutaneous veins very large. Stomach negative. Liver does not extend below the costal margin. Spleen cannot be percussed. Stools quite regular.

March 11.—Blood examination:

Red cells .....	4,340,000
White cells .....	11,625
Hemoglobin .....	70 per cent.
Globular value .....	0.84

March 12.—Examination of larynx. Posterior wall of œsophagus is pushed forward. Larynx totally deviated to the left. Paresis of right vocal cord.

March 13.—Ptosis of right eye-lid with external strabismus of the right globe. Edema of face and lids, and all the thoracic wall, especially on the right side. The tumor in the neck seems to have slightly decreased in size.

March 16.—A large, hard, painful mass of lymph-nodes can be palpated in the right axilla. Edema of right side of thorax still exists. Subcutaneous veins greatly dilated.

Patient hums incessantly and when asked why he does so, he replies that it is in spite of himself.

Strabismus of right eye still present.

March 23.—Restless, incessant cough during night. Lymph-node mass in right axilla is no longer painful, but on the other hand, painful glands are found in the left axilla. The tumor in the neck has decreased in size.

March 30.—Edema of prepuce, œdema and cyanosis of face. Temperature 102°F.

Blood examination:

Red cells .....	3,968,000
White cells .....	7,750
Hemoglobin .....	60 per cent.
Globular value .....	0.78

*Radiography* (see Fig. 8).

April 2.—Great dyspnœa. Neck, face, head very œdematous. External genitals the size of a child's head. No œdema of lower limbs.

April 4.—Face still more swollen. Eyes completely hidden by œdema of the lids. Great dyspnœa, some delirium. Pulse small,

cannot be counted. Cardiac sounds very weak, embryocardia. Death on April 5.

*Autopsy.*—Medium sized male, skin pale, œdema of face, neck, scrotum and penis. No œdema of lower limbs. Lymph-nodes of neck and axillæ tumefied.

*Abdomen.*—Great distension of intestine, especially of colon. Omentum poor in fat. Appendix and cæcum normal. About 20 c.c. of whitish, cloudy fluid in abdomen.

Liver extends one finger's breadth below costal margin. Spleen reaches the left costal margin. On the left the diaphragm reaches the fourth rib, on the right the fourth intercostal space.

*Thorax.*—A large tumor with a lobular surface and rather soft consistency almost completely fills the anterior portion of the upper part of the thorax, pushing both lungs backwards and the heart downwards and outwards.

The left pleural cavity contains 800 c.c. of light lemon-colored fluid. No adhesions. On the right there are a few small adhesions, but no fluid.

The organs of the thorax are removed together with those of the neck.

*Œsophagus.*—Mucosa pale. Some areas of thickening in the form of patches (?).

*Trachea.*—Slightly flattened from in front backwards by the tumor. Hyperæmia of mucosa.

*Bronchi.*—Somewhat dilated. Much mucus on the surface of the mucosa of the larger bronchi.

*Left Lung.*—Lower lobe free, with atelectasis. Upper lobe adherent to the tumor in front, with emphysema.

*Right Lung.*—Lower and middle lobes adherent to tumor, with atelectasis. Upper lobe free and emphysematous.

*Pericardium.*—Infiltrated by the neoplasm, especially in the neighborhood of the large vessels. Contains 40 c.c. of slightly cloudy lemon-colored fluid.

*Heart.*—Valves normal. Left ventricle, 11 mm.; right ventricle, 3.5 mm. Musculature firm, brownish in color.

A neoplastic thrombus in the vena cava completely occluding the lumen and adherent to the walls. Above the thrombus, which

measures 35 mm. in length, is an ordinary thrombus extending up to the brachiocephalic trunk and into the axillary vein.

The primary tumor, which measures 17 x 14 x 8.5 cm. exactly, occupies the site of the thymus. Its upper edge reaches 5 cm. below the thyroid and is situated exactly in the middle line.

The surface of the tumor is lobulate. On section it is also lobulate, pinkish white and soft in spots.

The enlarged cervical lymph-nodes are pink on section.

*Spleen.*—Measures 11 x 6 x 3 cm. Surface smooth, consistency firm. On section the parenchyma is reddish.

*Left Kidney.*—Measures 11 x 4 x 2.5 cm. Capsule easily stripped off. On section, parenchyma reddish. Pyramids not very distinct. Vessels of pelvis slightly injected. Suprarenal apparently normal.

*Right Kidney.*—Measures 12 x 4 x 3 cm. Same condition as left kidney.

*Pancreas.*—Apparently normal.

*Stomach.*—A few hemorrhages in the mucosa.

*Duodenum.*—Follicles tumefied.

*Liver.*—Measures 23 x 13 x 8.5 cm. Surface smooth, consistency firm. On section, parenchyma cloudy, slightly nutmeg from stasis. Choledochus patent. Gall-bladder normal.

*Bladder.*—Hyperæmia of mucosa. Contents slightly cloudy.

*Prostate, seminal vesicles and testicles* normal.

*Intestine.*—Some punctiform hemorrhages.

*Rectum.*—Hyperæmia and œdema of mucosa. Hemorrhoidal veins varicose.

*Inferior vena cava, portal vein, iliac, spermatic and femoral veins* normal.

*Spine.*—Marked scoliosis, otherwise normal on section.

*Microscopical Examination* (Prof. Askanazy).—The neoplastic cells form numerous masses situated closely together and do not appear to be separated by any particular fundamental tissue. However, in spots small vascularized fibrous trabeculæ can be detected.

The neoplastic cells are polymorphous, larger than lymphocytes and have a very visible protoplasm and a large vesicular nucleus.

A relatively considerable number of cells are undergoing mitosis. A few small areas of necrosis are also present.

The general structure of the neoplasm is that of sarcoma. The presence of cell groups without fundamental substance cannot change this diagnosis as the classic stroma of carcinoma is totally absent.

No elements of the thymus or eosinophiles are visible. The question of lymphogranuloma can therefore be excluded.

*Diagnosis.*—Large cell sarcoma.

*Summary.*—In this case we have a male, twenty-seven years of age, who, some six months before death, presented signs of marked compression of the thoracic organs, *viz.*, cough, dyspnœa, a very apparent network of the subcutaneous thoracic veins and dullness over the sternum.

A positive Wassermann temporarily led the diagnosis astray, leading one to suppose that the case was either an acquired or congenital syphilitic mediastinitis.

The regularly progressive progress of the symptoms of some intra-thoracic obstacle soon revealed the malignant nature of the process and made the diagnosis of a mediastinal tumor a certainty.

Besides, about one month before death, there was a deviation of the larynx to the right and paresis of the right vocal cord, ptosis of the right upper lid, right external strabismus of the eye and finally œdema of the lids, face and thorax. Death ensued with an enormous œdema of the upper part of the body, the lower limbs remaining intact.

At autopsy, a very large nodular, soft tumor, whitish on section, seated in the anterior mediastinum and region of the thymus, was found, pushing back the lungs and the heart, penetrating the pericardium and superior vena cava and compressing the trachea.

Microscopically, numerous masses of polymorphous neoplastic cells were found in the midst of a trifling connective tissue stroma. No elements of the thymus or eosinophiles could be detected so that the diagnosis of large cell sarcoma was made.

CASE VI.—Female, *æt.* 38 years, entered hospital September 21, 1907. Hereditary and personal antecedents negative. Patient states that she has always enjoyed excellent health.

*Present Illness.*—Patient thinks that the onset of the affection

began in May, 1907, although she had had a cough since the month of February, but supposed that it was due to a commencing gestation.

It was unquestionably in May that the cough increased, although there was no expectoration or thoracic pain. A physician consulted at this time found nothing abnormal, and the patient continued her occupation until the end of June.

At about this time she began to lose strength, coughed more, but with little expectoration. Another physician was consulted and told the patient that there was "no cause for worry." Nevertheless she was obliged to remain in bed during July. A very violent paroxysmal cough frequently occurred, especially when the patient was lying down, so that she was obliged to sit up in bed.

There was also a sort of tickling sensation in the throat, especially on the right, and this was the cause of the paroxysms of cough.

There had never been any difficulty in swallowing nor aphonia. In July, food was vomited immediately after meals, but the appetite was good and the stools normal.

On August 9, the patient had an easy labor at term, lost very little blood and got up five days later. Now, it was since the labor that the patient's condition really became aggravated.

Strength was progressively decreasing, there was loss of appetite and she lost several kilogrammes in weight. She could not work or nurse the baby. The patient did not remain in bed because the cough was worse when lying down. At this time also the body "became covered with pimples" (?) and a lymph-node developed in the left axilla.

In September, lymph-nodes were detected in the neck. The paroxysms of coughing became more and more frequent, but there was very little expectoration. At the beginning of September, she consulted a professor at Lausanne, who suspected the trouble was due to the lymph-nodes, as he found no pulmonary lesion.

About ten days before entering hospital, the patient's face was swollen in the morning, but this subsided by night-time. The phenomenon was temporary, only lasting a few days.

In the middle of September, the patient again consulted her physician, who referred her to hospital with the diagnosis of neoplasm (?) of anterior mediastinum.

*General Condition.*—Nutrition fairly good, not very emaciated, but looks worn and distressed.

There are numerous excoriations and lesions from scratching on the back, flanks, external aspect of the upper limbs and anterior aspect of the legs.

The patient was seen by a dermatologist who diagnosed a toxic, prurigo.

*Lymph-nodes.*—At the anterior wall of the left axilla there is a large, movable, painless lymph-node the size of a hen's small egg and very hard to the feel. The skin covering it is absolutely normal.

In the left supra-clavicular fossa quite high up near the posterior edge of the sternomastoid can be felt another lymph-node the size of a pigeon's egg. Lower down, directly above the clavicle is a third lymph-node the size of a bean.

In the right axilla and supra-clavicular fossa three or four small lymph-nodes the size of a pea can be palpated. When the patient coughs some small lymph-nodes can be detected in the right supra-clavicular fossa which appear to be situated more deeply. All these lymph-nodes are movable, hard and painless.

Nothing to note in the inguinal regions. No œdema of face or lower limbs.

*Respiratory System.*—Thorax deformed and very asymmetrical. The right hemithorax presents a marked vaulting, especially pronounced in the antero-posterior diameter. At the junction of the upper with the lower two-thirds of the sternum there exists a marked forward convexity.

*Lungs.*—Behind there is slight dullness of the apices over the supra and infra-spinous fossæ, especially marked on the right. Resonance normal in the mid-region and bases. Exaggerated vibrations on both sides.

By auscultation there is an extremely whistling respiration, especially at expiration, at the apices in the supra-spinous fossæ. This expiratory whistle, which is intense and rough, is about equally marked in both apices. It also extends lower down in the infra-spinous fossæ, but its tone is softer.

In the mid-pulmonary region and at the bases, the breathing

can be distinctly heard. Bronchophonia in the supra and infra-spinous fossæ. Complete absence of râles everywhere.

In front, dullness was quite marked in the right supra-clavicular fossa. Below the clavicle and over the entire right lung dullness was absolute and compact, becoming confounded with that of the liver. The vocal vibrations were exaggerated throughout the right lung. Bronchophony.

On auscultation, whistling respiration, especially marked in expiration. The same rough expiratory souffle existing behind was present in front, with a maximum in the infra-clavicular fossa, but was present over the entire lung. Some sibilant râles.

The resonance was normal in the left lung. Respiration very distinct. Propagation of the souffle in the right lung.

The patient suffers almost continually from paroxysms of very dry cough, especially when in the recumbent position. A small amount of greenish mucopurulent expectoration.

*Circulatory System.*—No cardiac shock or vibrations perceptible. The apex of the heart cannot be distinctly made out. The heart sounds were regular, a little distant, and almost concealed at the base by the respiration. No souffles. The radial pulses are isochronous and equal. Pulse full, ample, regular at 98.

*Digestive System.*—Fairly good dentition. Tongue clean and moist. Abdomen soft. Diastasis of recti muscles. No spontaneous pain, palpation painless.

The liver dullness extends four fingers' breadth below the costal margin. The edge of the liver can be easily palpated. Boundaries of stomach normal. Spleen normal, can be percussed, but not palpated.

Appetite diminished. Stools normal.

*Nervous System.*—Normal intellect. Pupils somewhat contracted, perfectly equal, reacting well to light and accommodation. Patellar reflexes normal. Cutaneous plantar reflexes weak. Babinski and clonus absent. Sensibility and motility normal.

*Urogenital System.*—Menstruated at eighteen, always regular and normal. Menses have not occurred since the last labor.

Urine clear, yellow, no sediment. Albumin, sugar and urobilin absent.

September 27.—Dullness in the mid-portion of right lung behind.

In front on the right, compact dullness over the entire thorax.

These signs are probably due to a tumor of the mediastinum pushing aside and compressing the lung, and is almost certainly a lymphosarcoma of the mediastinal lymph-nodes.

The evolution of the affection indicates that there have been no pulmonary phenomena other than those resulting from compression by the tumor (Professor Bard).

September 28.—Blood examination:

Red cells .....	3,658,000
White cells .....	9,200
Hemoglobin (Gowers) .....	80 per cent.
Globular value .....	1.09
Polynuclears .....	70 per cent.
Mononuclears .....	30 per cent.

Red cells are normal in shape, isocysts.

September 30.—Condition stationary. Violent paroxysms of coughing in spite of large doses of codeine.

Examination of larynx, absolutely normal.

October 7.—No change other than more frequent paroxysms of coughing. The temperature, which was very irregular, averaged about 100.8°F.

October 10.—Exhaustion progressing. Orthopnœa increasing and persists during the night in spite of morphine. The paroxysms of coughing becoming more and more frequent, sometimes subin-  
trant, end by expectoration of rather copious mucopurulent sputum.

In the right lung behind there is marked dullness over the apex and mid-pulmonary region. Dullness over left apex.

By auscultation a focus of medium and large moist râles could be detected in the middle of the right lung. Whistling respiration still present without any change.

In front, the dullness was compact from the clavicle to the base of the lung and became confounded with that of the liver and extended beyond the left edge of the sternum also becoming confounded with the cardiac dullness which itself extends to the sixth intercostal space. There is the same intense souffle as in the right lung. In front, the left apex remains resonant.

The heart sounds are still distant, well struck, but rapid. Pulse, 120.

No new enlarged lymph-nodes.

October 14.—Symptoms progressively increasing in intensity. The orthopnœa is still more distressing and with difficulty influenced by morphine even in large doses. Pulse regular at 120.

October 15.—Ophthalmological examination negative.

October 16.—Death.

*Autopsy* (Professor Askanazy).—Medium sized female. Skin covered with small raised blackish spots, especially on the limbs and trunk. Nipples deeply pigmented. Abdomen not distended. Subcutaneous fat poorly developed. Slight malleolar œdema.

The liver extends the breadth of a hand below the costal margin. The spleen does not reach below the ribs.

A little serous fluid in pelvis. Atrophy of omentum. Transverse colon at level of umbilicus.

The diaphragm, above which a very hard resistance existed, extends on the right side to the fifth intercostal space and on the left to the fifth rib.

When dividing the costal cartilages neoplastic masses are seen in the left pleural cavity and anterior mediastinum.

A voluminous tumor (see Fig. 4), strongly adherent to the posterior surface of the sternum, especially at the level of the insertion of the second and third left ribs, is exposed. Besides, there are hard, whitish neoplastic patches and nuclei in the anterior mediastinum, as well as indurated lymph-nodes, some of which are the size of a hazel-nut. These lymph-nodes follow the mammary vessels on the left side.

On the right side of the neck a few lymph-nodes exist along the large vessels. There are also a few small indurated glands in both axillæ.

In the left pleural cavity were about 300 c.c. of pink, slightly cloudy serous fluid. Some loose fibrous adhesions unite the left lung with the parietal pleura.

On the right side the pleural cavity is totally obliterated.

The pericardium could not be seen as it was completely covered by the tumor. There is about 100 c.c. of lemon-yellow fluid in the pericardium containing a few shreds of fibrin.

On the left side the neoplasm has commenced to penetrate the pericardium.

Very marked œdema of the mediastinal cellular tissue.

Heart slightly dilated. Adipose tissue exists. Much coagulated blood in the cavities. Muscle quite firm and brownish red in color. Valves intact.

The tumor, whose site corresponds to about that of the thymus, is compact and extends from one side to the other of the thorax in the upper part of the anterior mediastinum. The tumor and both lungs were removed *en masse*.

The measurements of the neoplasm are: From left to right, 13 centimetres; vertically, 8 centimetres; antero-posterior diameter, 9 centimetres.

The parenchyma of the neoplasm, whitish in color, is rather hard and elastic and grossly lobular in structure in certain parts.

The growth surrounds the superior vena cava, whose posterior wall is pushed forward, presenting a slightly convex surface.

At the point of anastomosis of the azygos vein, the neoplasm has perforated the walls of the vessel and forms in its lumen a projection the size of a bean with a smooth surface.

The left brachiocephalic vein is flattened, but does not contain neoplastic tissue. The pulmonary veins on the right present several neoplastic nodules on their internal aspect.

At the level of the hilum, a small neoplastic nodule is present on the pericardium near the pulmonary vein.

The œsophagus is intact. On the contrary, there are some neoplastic patches on the walls of the trachea and right bronchus.

In the posterior mediastinum only a few lymph-node foci are seen on each side of the trachea. Some of them reach down to the diaphragm. The prevertebral tissue is intact.

The left lung presents some fibrous adhesions and a certain number of small whitish patches under the pleura of the upper lobe. In the lower lobe is a neoplastic nodule the size of a walnut, formed by several agglomerated whitish nodules.

The right lung, which is very large and heavy, offers, on section, a notably different aspect between its anterior and posterior portions. In the posterior portion of the upper lobe, the parenchyma is invaded by very numerous grayish nodules. These are separated from each

other by a pinkish or yellowish pulmonary tissue; they vary in size from that of a pin's head to a bean. They are less numerous in the lower lobe than in the middle and upper lobes.

In the anterior portion the pulmonary parenchyma presents, on the contrary, a neoplastic infiltration almost diffuse in nature. A small amount of yellowish tissue alone indicates the remains of the parenchyma, and the slightly dilated bronchi are entirely occluded by an amber-yellow gelatinous mass.

The lymph-nodes of the hilum are anthracotic and contain numerous neoplastic nodules. There are also some metastatic nodules on the left parietal pleura. A chain of enlarged lymph-nodes, containing neoplastic masses, follows the aorta down to the pelvis.

The mesenteric veins are very injected. Some fibrous adhesions around the appendix.

Spleen increased in size, measuring 13 x 10.5 x 4 centimetres. Through the tense capsule one can perceive several white nodules which, on section, offer the same characters as the neoplasm.

The fatty capsule of the left kidney is atrophied. Its fibrous capsule is easily stripped. The surface of the kidney is slightly granular. The kidney itself is somewhat indurated, its cortical substance is prominent and atrophied and the glomerulæ pale. The organ is normal in size.

The right kidney presents the same changes as the left and is also normal in size.

Both suprarenals appear to be normal. Their medullary substance is well developed.

The stomach contains a little grayish fluid. The mucosa is covered by a layer of mucus and shows the beginning of gastromalacia.

The liver is the seat of pronounced stasis, especially in the right lobe, and presents some atrophy of the lobular centres. Considerable œdema of the gall-bladder.

The intestinal mucosa, strongly injected presents punctiform hemorrhages in all its folds. The hyperæmia extends throughout the large intestine and becomes attenuated in the descending colon. No notable tumefaction of the lymphatic system.

The mucosa of the bladder is injected.

The skull is somewhat sclerosed. The dura is very tense, the meninges very pale. Corpus callosum somewhat prominent.

The thin cerebral arteries contain little blood. The fluid in both lateral ventricles is increased. The choroid plexuses are rather pale. Cerebral substance normal.

*Microscopic Examination* (Professor Askanazy).—The major part of the tumor is composed of cells whose size and shape correspond nearly to those of small lymphocytes. These cells are in a tissue not presenting the structure of a typical reticulum, but rather that of loose connective tissue. However, this tissue becomes condensed in certain areas forming a sclerosed connective tissue in which the lymphocytes form masses more or less close together.

Besides the lymphocytic dissemination, two other important histological phenomena are to be noted: On the one hand, the existence of large cells in the midst of the neoplastic tissue; on the other, the presence of cell masses in the interior of the lymphatic vessels.

The large cells are in places relatively numerous, at other spots, on the contrary, they are completely isolated, situated in the clefts in the surrounding connective tissue.

These cells, rounded in shape, rarely elongated, possess a voluminous protoplasm containing one or several nuclei with large nucleoli. The nuclei are often near the surface of the cell and their nucleoli sometimes offer a diameter greater than a red blood-corpuscle.

In none of the preparations was it possible to distinguish any elements originating from the thymus. No eosinophiles or cells with large nuclei (Sternberg) are visible.

*Summary.*—In this case we are dealing with a female, thirty-eight years of age, in whom distinct signs of an intrathoracic tumor appeared about six months before death.

At the onset there were symptoms of mild irritation, followed by more marked ones of compression of the respiratory tract and finally paroxysms of suffocation and orthopnoea.

A labor, four months after the appearance of the early signs of respiratory difficulty, seems to have hastened the evolution of the process. One month before death, cervical, supra-clavicular and axillary lymph-nodes developed, fleeting œdema of the face, then vaulting of the sternum and thorax accompanied by dense and very

extensive dullness throughout the right lung. Larynx negative. Never any thoracic pain.

Autopsy revealed the existence of a malignant neoplasm of the mediastinum occupying the entire upper part of the thorax—anterior mediastinum and region of the thymus—and invading the right lung, pericardium, superior vena cava, right pulmonary vein and trachea.

Microscopically, the growth was composed of masses of lymphocytes situated in the midst of a loose connective tissue with rather numerous large neoplastic cells. No eosinophiles or elements of the thymus could be detected. The diagnosis was lymphosarcoma of the anterior mediastinum.

#### REMARKS

*Signs at the Onset.*—The symptoms arising in the early phases of development of malignant tumors of the mediastinum are particularly important. In my cases these symptoms offered certain characters that I will consider.

*Respiratory System.*—In five of the cases *cough* was the first symptom, sometimes the only sign verified at the onset. It was usually a hard and frequent cough, sometimes irritable and paroxysmal, more often progressively increasing and particularly stubborn to all treatment essayed.

The *dyspnœa*, on the contrary, was not common at the onset. In only two cases (Cases 3 and 4) did it develop early in the evolution of the process and both cases rapidly evolved to a fatal issue leading one to suspect that perhaps the process was in reality more advanced than the case histories might lead one to suppose.

Only one of the cases (Case 3) presented early laryngeal phenomena, the essential symptom being dysphonia, although in these rapidly evolving cases some lesion of the vocal cords has been discovered by the laryngoscope.

*Expectoration*, which is frequently noted in the advanced phases of the process, is, on the contrary, absent in the early stages.

As to the signs revealed by pulmonary auscultation at the onset they were found in three cases. In Case 1, there was dullness over the sternum; in Cases 2 and 5, there was considerable dullness over one apex.

*Circulatory System.*—This system also gives rise to few signs at the onset. In two patients (Cases 4 and 5) there was an early development of a *collateral circulation* in the form of a subcutaneous venous network over the thorax. In the case of a twelve-year-old child (Case 2) repeated *epistaxis* took place.

*Lymph-nodes.*—Frequently considered as one of the signs revealing intrathoracic malignant tumors, enlarged glands occurred in only two of my cases (Cases 2 and 4) at the onset. In both cases the cervical and supra-clavicular lymph-nodes were the seat of tumefaction.

*Digestive System.*—In only one case were there any early symptoms in the digestive tract. This occurred in Case 4 and consisted of *difficulty in deglutition* and later on by vomiting of food immediately after meals.

*Nervous System.*—This system furnished few symptoms at the onset. One patient (Case 4) complained of early painful symptoms due to compression of the nerve trunks by the neoplasm. Another patient (Case 3) suffered from headache which was increased by cold.

The *urogenital system* never gave rise to any symptoms at the onset.

*General Symptoms.*—These were not important during the early phases of the process and only occurred in two patients (Cases 2 and 4) in the form of *slight emaciation* and *loss of strength*. In the other four patients the general health remained normal for quite a long time.

*Signs Appearing during the Later Phases of the Evolution of the Neoplasm.*—In all my six cases there was an aggravation of the symptoms present at the onset resulting from the gradual increase in size of the intrathoracic growth.

In the *respiratory system* the *cough* increased, likewise the *dyspnoea*. The signs found by auscultation extended and complications developed in the domain of the respiratory tract, such as a dry or exudative pleurisy (Cases 2 and 4), *hemoptysis* (Case 4), displacement of the larynx (Cases 1 and 5), *dysphonia* (Cases 1, 3 and 5), *orthopnoea* (Cases 1, 2, 3 and 6), *paroxysms of suffocation* (Cases 1, 3 and 6) and slowly developing deformity of the thorax (Cases 1, 4 and 6).

The progressive increase in the size of the growth made itself

felt in the *circulatory system*. *Cyanosis* of the face, sometimes of the hands and nails as well, on one or both sides, is a common symptom in my cases (Cases 1, 2, 3 and 5). The cyanosis usually remained localized to the face and was not accompanied by any notable oedema.

However, in Case 5 there was puffing of the face with extensive oedema over the thorax and external genitals although there was no oedema of the lower limbs.

Oedema without cyanosis occurred in one case (Case 4), and was localized in the left upper limb. Repeated epistaxis occurred in two patients, both children (Cases 1 and 2).

In four of my patients the deep circulation, which at first was interfered with, became reestablished by the development of a more or less important subcutaneous venous network. This collateral circulation usually developed in the thorax; less frequently in the abdomen (Cases 1, 3, 4 and 5).

*Displacement of the heart* was met with in two patients and in both it was towards the right side (Cases 1 and 4. See Figs. 2, 5 and 6).

The displacement was not accompanied by more marked circulatory disturbances than those occurring in cases of mediastinal tumors in which the heart remained in its normal site.

The *cardiac rhythm* did not appear to be greatly influenced excepting in Case 5 in which embryocardia was noted. On the other hand, the frequency of the pulse rate was increased although the tachycardia was never excessive, the pulse rate being on an average of 120. The blood-pressure offered nothing of interest, although it must be said no particular notes in this respect are to be found in the case histories.

Few important disturbances of the *digestive system* were found. Dysphagia, especially for solid food, is mentioned in two cases; once it was very marked (Case 4) with an early onset and with a slow progressive increase. On the contrary, in Case 1, it occurred late in the process and was mild.

The abdominal viscera offered nothing of interest during the evolution of the thoracic tumor.

The *nervous system* gave rise to various painful phenomena,

*thoracic pain* in particular. Only one case presented serious nervous disturbances characterized by unequal pupils, sluggish reflexes to light and accommodation, strabismus, palpebral ptosis and weak patellar reflexes (Case 5).

There was nothing particular to note in the *urogenital system*. In spite of renal metastases, occasionally numerous, the urine was always normal.

During the evolution of the tumor, metastases in the cervical, supra-clavicular and axillary lymph-nodes were present in all the six cases.

*Terminal Symptoms*.—In all the cases these were quite similar. In the foreground was *dyspnœa*, regularly and progressively increasing, ending in orthopnœa and paroxysms of suffocation. Orthopnœa occurred in four cases and attacks of suffocation in three.

In Case 1 only, were there any really important changes in the larynx toward the end of the process made evident by dysphonia. Laryngoscopy showed a backward displacement *in toto* of the larynx with no change of the vocal cords other than a slight injection of the mucosa.

Cases 4 and 5 are remarkable in that they never presented any of the serious disturbances of the respiratory tract usually encountered in these circumstances.

In Case 5, the disturbances of the venous and lymphatic circulation in the upper half of the body soon became preponderant. The patient soon died with cyanosis and especially considerable œdema of the head and trunk. No trace of œdema of the lower limbs.

As to *treatment*, there is little to be said. Radiotherapy was employed in Cases 1, 4 and 5. Very badly tolerated by Case 1—a child of five years—it seems to have had a fleeting favorable influence on one patient (Case 4), but no conclusion can be made in this respect in Case 5, because the lapse of time during which the X-rays were used was much too short.

*Iodine, arsenic* and *Hg*, no matter in what form they have been exhibited, have never been known to exercise any notable influence on the evolution of the process and without exception the therapeutic results obtained have been extraordinarily bad.

## CONCLUSIONS

I. Primary malignant tumors of the anterior mediastinum and region of the thymus originate from different tissues and organs contained in the mediastinum. The anatomical structure of these neoplasms is known and generally easily recognized.

II. The thymic origin of the majority of primary malignant tumors of the anterior mediastinum usually cannot be demonstrated.

III. The term "malignant growths of the thymus" should be reserved, until further light has been thrown on the subject, to tumors of the mediastinum clearly and unmistakably containing anatomical elements derived from the thymus, *viz.*, Hassal's corpuscles and cortical or medullary cells of the thymus.

# Pædiatrics

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## OSTEOMALACIA ESPECIALLY IN CHILDHOOD

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THE mere fact that, for a disease such as osteomalacia, attempts are still being made to discover the cause, and that many factors are invoked to explain its onset, amply implies that up to the present time we are as yet in the dark. In fact, if in adults various influences have been supposed to produce osteomalacia, there is all the more reason that in the case of children some of these must be eliminated from the etiology and that others should be added. At any rate it may be said that:

(1) Osteomalacia, as a morbid entity, is far less common in children than in adults;

(2) That if various statistics be taken into consideration, this morbid process varies in different countries, hence a kind of endemicity exists in certain regions. The very fact that a more or less regular geographical distribution of osteomalacia exists implies that the affection must be influenced by climate and soil.

In point of fact, it is encountered endemically in damp valleys and marshy districts. It is endemic in the district of Bâle (Fehling), in Alsace and the Black Forest, in the country around Heidelberg (Rehrer) and Baden. Gelpke, who has studied the distribution of this disease, points out that the different forms of osteomalacia are not equally frequent in the same country. This observer was the first to consult the death statistics of osteomalacia and believes to have found 89 cases in childhood, but an interesting point to note is that having consulted the Italian death statistics from 1881 to 1886, he found out of a total of 7,073,600 deaths there were 89 cases of infantile osteomalacia, while in the Swiss statistics from 1880 to 1888 there were 92 cases of osteomalacia in adults, in the statistics of Bavaria from 1884 to 1887 there were 73 cases and finally the Registrar General of Births for 1875 to 1880 gave 96 cases. But in the three latter statistics all the deaths occurred in

adults, both male and female, and no mention of osteomalacia in children is made.

What conclusion can be drawn from these data? It is probable that in certain countries where one was not familiar with this morbid entity evolving in children, it was not recognized even if it did exist and probably those cases which did exist were classed under some heading other than osteomalacia; and inversely, where this disease was recognized its prevalence was exaggerated.

What would be interesting to discover, given the lesions, would be the quantity of lime and the quantity of acid contained in the waters of various countries. And this leads me to say a few words in respect to the influence of water on the appearance and evolution of this disease.

Unquestionably the influence of water is not an all-important one in respect to osteomalacia evolving in very young children such as some cases I shall refer to, but on the other hand it is important in cases of older children. Gelpke carried out his researches in this direction in the Ergolz valley where he was able to collect nineteen cases of osteomalacia, but he could not come to any satisfactory conclusion. According to the results obtained, more lime was found in the water of this region than in others. On the other hand, Cossati found that the water of the valley of Olona, which was rain-water that the inhabitants were obliged to collect because of the too great depths of the wells, was deprived of lime salts.

*The influence of food* is a question of prime importance as an etiological factor of osteomalacia. In point of fact, in many cases of this disease it will be noted that the child had been breast-fed, but on the other hand only in a case recorded by Kissel is any mention made of the mother's health, this point being passed over in silence in all others, and it is just this cause that some writers evoke by comparing osteomalacia to rickets, especially from the viewpoint of etiology.

If one goes over the case histories of a large number of children with osteomalacia, it will be found that most of them were reared on mixed feeding—breast and bottle—and that afterward the diet was defective, facts which should lead one to suspect that feeding plays a large part in the development and especially the evolution of the

disease. On the other hand, when several children of the same family are fed in about the same way and only one develops osteomalacia one may conclude that feeding has a large share in the eclosion and evolution of the disease but that association of other etiological and pathological conditions is absolutely necessary.

If we consult the various publications on this subject it at once becomes evident that opinions differ as much on the question of alimentation in general as on each article of diet in particular. Thus Gelpke believes that it is the want of meat that provokes the disease; on the contrary, Kehrer denies this by stating that the majority of cases of osteomalacia are met with in the wives of butchers and bakers. During the siege of Paris, Charcot observed four osteomalacic females who died at the Salpêtrière, but never before had he met with so many cases in such a short lapse of time, and he supposed that the disease was induced by all the privations resulting from the siege.

*The Influence of Damp Dwellings.*—Myatowitch, in 1875, stated that osteomalacia was a disease of the working classes and attributed it to cold and damp dwellings. Fehling believes that the effect of dampness is doubtful; while Kehrer maintains that the disease is not confined to the poor. In the cases he has been able to collect, Meslaye states that some of the patients had lived for some time in damp places, but that this influence was completely absent in others. Finally, Rosier points out that it is difficult to discuss this subject knowing that from the viewpoint of climate and dampness that of Calabria and South Germany are very different and still these are the two centres *par excellence* of osteomalacia.

As to *the influence of heredity* there is little to say as the divergence of opinion is very great in this respect. From all that has been published on the subject one may suppose that heredity as an etiological factor is very uncertain. In this respect Eckmann's case is often referred to. It was that of a Norwegian family in which deformity of the bones continued through three generations but Collineau is inclined to believe that this was an instance of rickets and not true osteomalacia. Fehling has reported the case of a brother and sister who were both afflicted with osteomalacia and I would add the case of L. Tixier and Broca, of two sisters presenting the disease.

*The influence of age* is unquestionable; osteomalacia is above all an affection of adult age. But this rule is not absolute as there is an infantile osteomalacia, just as there is a senile osteomalacia. It is very rare in nurslings but is not infrequent in children from the age of fifteen years as the following table, due to Stansky, shows:

Nursling .....	1 case
16 to 30 years .....	8 cases
30 to 40 years .....	6 cases
40 to 50 years .....	3 cases
50 to 60 years .....	4 cases
Above 60 years .....	2 cases

All of Polgar's patients were between the ages of twenty-eight and thirty-eight years, and of the sixty-six cases recorded by Eissenhart the youngest patient was eighteen, the oldest forty-five. Hensel's eight patients varied in age from eighteen to fifty-one years, while in twenty-nine cases reported by Reybard two patients were under twenty years of age.

*The Influence of Sex.*—All observers agree that osteomalacia is of extreme frequency in women while it is rather rare in men. For example, in a total of 135 cases reported by Litzmann, eleven were males; Mayolin's statistics give a proportion of 20 females to one male, while Bouley's statistics give a proportion of three females to one male.

But these statistics have no bearing on the influence of sex in children, because as I have pointed out the etiological conditions vary according to whether one is dealing with osteomalacia in adults or children. In point of fact, in cases of this disease in adults, one must take into consideration as an etiological factor the influence of pregnancy, the post-partum, multiparity and nursing. If various statistics are consulted it will at once become clear that all these causes are far from negligible, and of course cannot be applied to cases of osteomalacia in children.

The demonstration of infantile osteomalacia itself discards the etiological factor so frequently invoked in the case of adult females, namely, the ovarian theory. In the case of little girls the ovaries being in a rudimentary state, they cannot, either by an exaggerated or insufficient internal secretion act in one way or another on bone

decalcification. Hence the ovarian theory of osteomalacia, maintained by some, is devoid of value in female children.

In the following case due to Bérard and Normand, of Lyons, the father and mother of the patient were syphilitic.

Female, *æt.* 7 years, entered hospital on August 26, 1903.

*Hereditary Antecedents.*—Mother well, had been married twice. Had two healthy children. Later on the husband became alcoholic and contracted syphilis, after which the patient was born. The wife must have been infected, because with her second husband—the present one—she has had two miscarriages. No case of osteomalacia in the family.

*Personal Antecedents.*—Walked at the age of ten months; dentition tardy but regular.

Measles at the age of four, bronchitis six months later. From this time on there were digestive disturbances lasting for several years with recurring attacks of diarrhoea and vomiting.

The patient, who at the age of one year was walking well, began six months later to complain of tired legs and did not want to walk. At about the age of four pain in the lower limbs was complained of, the child no longer being willing to walk and suffered both day and night as well.

At the age of five the legs were distorted, the pain became progressively more acute, was spontaneous and exaggerated by movement. At this time the lower limbs alone were involved.

The child was admitted to hospital for a strangulated prolapse of the rectum on August 26, 1903, for which she was operated on at once.

Very marked incurvation of the tibiae and fore-arms was noted, and suspecting that these deformities were the result of rickets several manual osteoclasis were done. Correction of the deformities was easily obtained and the limbs immobilized in plaster casts. An elongation of four centimetres was thus obtained.

In October the first cast was removed when it was found that there was total softening of the lower limbs and complete absence of consolidation of the osteoclasis and a tendency to incurvation of the tibiae and femurs.

The patient was left a month without any plaster casts, during

which time she suffered very severe pain not only in the lower limbs but in the upper limbs and pelvis as well. The deformity of the legs became more marked and the fore-arms presented a strong incurvation.

November, 1903.—The limbs were straightened and put up in plaster under narcosis. Cod-liver oil and calcium lactophosphate were prescribed.

December, 1903.—At the end of a month the casts were removed; the osteoclasias had not united and all the bones were soft. For twelve days a 1:1000 solution of phosphorus in oil was exhibited at the daily dose of two drops, but it could not be continued on account of the resulting diarrhœa and vomiting. The child was immobilized in a large plaster cast leaving the limbs exposed to the air.

January 3, 1904.—At the end of a month the bones were quite as soft as before. Ext. thyroid at the dose of ten centigrammes per day was essayed, but this medication also produced diarrhœa.

February 19, 1904.—No trace of bone consolidation. The bones of the legs and thighs were like paste, while softening of the upper limbs increased and movement caused pain. A posterior plaster cast was made using Bonnet's wire body splint as a frame. Cod-liver oil for three months; ext. thymus for a fortnight; no amelioration.

June 10, 1904.—The child no longer suffers. A mixed Hg. and iodide treatment was given for a fortnight but had to be stopped on account of digestive disturbances. No improvement could be detected in so short a time.

While at rest the child did not suffer and was very quiet. No evidence of craniotabes. Although the intelligence was not very developed, it appeared to be intact. The digestive disturbances persisted and at times became aggravated. The urine, frequently examined, showed traces of albumin, lowered urea and phosphates.

There was no globular anæmia; the leucocyte count was normal. No eosinophilia. Temperature chart was normal. Radiography showed very transparent bones reduced to a thin osseous shell at certain points and generally atrophied.

October, 1914.—The child died quickly in a day or two presenting the phenomena of bronchopneumonia, such as she had had in August.

In spite of the negative attitude in respect to syphilis assumed by some observers, this infection, like the toxi-infections, may be a causal factor of osteomalacia. The case just given offered a long period of digestive disturbances—vomiting and diarrhœa—at the same time that the bone lesions developed. Now, was there not a relationship between the two morbid states, as has been admitted for rickets? The association of rickets or osteomalacia with syphilis can be understood, the digestive toxins being a cause in either of the two pathologic processes.

#### PATHOLOGY

The principal lesions of osteomalacia are in the bones, and in the same patient may offer different degrees according to the phase of the disease or the evolution of the affection. Thus a phase characterized by a change in the texture of the bone—phase of onset—is observed, then a phase of softening and lastly a phase of fragility. The two latter phases produce changes in the shape, consistency and continuity of the bones.

The change in the shape of the bones results both from modification of elasticity and resistance of the osseous tissue. From the onset the osseous structures became less compact and rarefied at the same time that it decreases in volume, while occasionally it softens to an extreme degree. It seems as if the bone were reduced to a fibrous periosteal mass surrounded by a pulpy tissue comparable to hepatic parenchyma. However, the bone may preserve a certain amount of resistance; the compact tissue is rarefied but a more or less thin shell remains of remarkable fragility, hence the frequency of spontaneous fractures. These result in new deformities due to a change in the continuity and the development of a very irregular callus with incomplete ossiform productions. The bone may collapse and, so to speak, sink down like a soft lump of wax or rubber, becoming broad, undulated and shortened. The epiphyses may be swollen, but in a more regular fashion than in rickets.

Therefore, *softening and fragility of the bones* are the two principal anatomical characters of osteomalacia, but usually, as I have said, fragility precedes the softening and in the majority of cases the first symptoms of the disease will be spontaneous fractures and this cardinal symptom clinches the diagnosis.

It was this division into two phases that led Kilian to admit two forms of osteomalacia—first phase or osteomalacia fracturosa, and second phase or osteomalacia flexibilis.

The bone softens and can easily be cut with a knife, then becomes flexible like wax, gorged with blood and quickly drying when exposed to the air. The deformity of the thorax observed in this disease is the result of osseous flexibility. As the lesions progress atrophy of the bone occurs and the skeleton undergoes a kind of atrophy *en masse*, as collapse of the framework of the body resulting in a decrease of the height of the patient.

All these changes of shape and conformation are present in variable degrees from one patient to another, and according to the parts of the skeleton involved. Thus deformities of the skull are usually less pronounced than in other parts of the skeleton. The skull retains its normal aspect but nevertheless offers a certain roundness; what predominates is softening of the bone with thickening, a spongy vascular condition of the diploë and disappearance of the sutures. In some cases only a peculiar thinness of the bones of the skull is observed. Finally, in others, on the contrary, there is an increase in the size of the cranial bones which may attain considerable dimensions as, for example, in the following case reported by Meslaye.

Female, *æt.* 17 years. The cranial portion of the head was strikingly large. The lower part of the face was unchanged, the lower jaw being normal in size. The frontal region was broadened and raised up and covered by a series of bosses and depressions. Palpation provoked intense pain and gave to the exploring finger the sensation of soft putty.

Deformities of the spine are very frequent and the most varied degrees of scoliosis and kyphosis are met with. They are the result of an exaggeration of the normal curves, the deviations being due to the position of the patient, the various muscular actions, pressure exercised by the limbs or neck, as well as the sinking of the vertebral bodies, and lastly, periosteal osseous productions.

However, in fractures the periosteal productions are, in the case of the spine, far less frequent than in the case of the limbs. Deformities due to an exaggeration of the normal curves of the spine offer a certain regularity. The dorsal region forms a more or less lateral

projection, the cervical region tends to become horizontal to such an extent that the chin may rest upon the sternum producing flattening; in the lumbar region a kind of saddle-back is formed, while the thorax and pelvis likewise participate in the changes.

*Macroscopical Changes.*—These are characteristic. The periosteum is ordinarily thickened, softened or œdematous; it is easily peeled off the bone, while the subperiosteal stratum sometimes forms a kind of pulp comparable to the marrow or the tissue of gingivitis. The insertions of the tendons are thickened, infiltrated when the periosteum has been removed, a thing that is sometimes so easy that strips may be peeled off which only adhere to the muscular insertions.

The osseous surface is spotted red; the little vascular depressions usually hardly visible are exaggerated forming small spots with a more pronounced vascularization. In a more advanced degree the osseous surface is rough as if granular and finally the bone tissue becomes irregularly spongy at the surface of the diaphysis and epiphysis.

Upon section of the bone these characters are more pronounced, but differ in the long and short bones. In the former, in the early phase, there is an exaggeration of the medullary canal and atrophy of the trabeculæ of the compact tissue, while at the same time these become friable and appear to have reached the state of cartilagification, that is to say, they affect the characters analogous to bones submitted to maceration in an acid solution or to fetal bone.

In the short bones and epiphyses the conditions vary; the marrow is abundant, but there is often a sinking in of the trabeculæ which does not prevent either fragility or flexibility.

These changes become much more marked as the process progresses. Medullization of the osseous tissue, that is to say, predominance of the medullary tissue becomes more and more evident. Sometimes it is fat or a kind of adipous tissue which fills the enormously enlarged medullary cavities which are also infiltrated, grayish or yellowish in color, at other times the medullary tissue is red and the marrow seems to have returned to the fetal type. Finally, the lesions become more marked, the bone may present a kind of cystic transformation, the mass of the diaphysis of a long bone like the tibia or femur presenting irregularly rounded medullary dilatations, red,

infiltrated with blood and fat, separated by thin bony trabeculæ, circumscribing irregular oval cavities recalling the aspect of osteosarcoma, myeloid tumors or bulbous osteitis.

From the macroscopical viewpoint the difference between osteomalacia and osteoporosis is sufficiently distinct so that confusion is impossible. In point of fact, while fractures in osteoporosis have a distinct tendency to undergo immediate consolidation with a large residual callus, in osteomalacia union takes place by pseudoarthrosis, the following case due to Dufour and Legras being an example:

Female, born at term, had a fracture of the right leg at the age of one year which at first was unrecognized. She was late in walking, only taking her first steps at the age of four years. First tooth was cut at two years. Menstruated at twelve years regularly and in normal amount.

The general health has always been satisfactory but the patient has had in all *twenty-three* fractures which have kept her in bed for a long time. Some consolidated very slowly with a callus, but a fracture of the humerus has never united. There fractures occurred from very slight traumata.

The first fracture occurred at the age of one year, involving the bones of the right leg at their lower third. The second fracture occurred at the age of seven in the site of the first fracture of the right leg and again at the age of nine it refractured. Two years later she fractured the left leg at the union of the middle with the lower third. Then she fractured the arm at the lower third of the diaphysis of the humerus. The ends were not coapted, union did not take place but absorption of the osseous tissue did occur.

The left arm in turn fractured and a callus formed; then the following year another trauma resulted in a second fracture which, this time, did not unite and absorption of bone took place instead.

Finally, fracture of the right thigh occurred which was treated by permanent extension and united by callus. Altogether, there were twenty-three fractures, but these did not behave in the same way in respect to their evolution. Some united without giving rise to much deformity, others developed a callus with deformity, still others resulted in union between two contiguous bones—the right tibia and fibula revealed by radioscopy. Lastly, there were others which did

not produce a callus, but union was effected by the interposition of more or less muscle. Radiography showed that there was decalcification of the skeleton.

*The Histology of Osteomalacia.*—The changes arising in the spinal cord differ. In some cases it is normal or red or yellow. The red color of the marrow was formerly supposed to be due to hyperæmia and Rindfleisch supposed that this was the cause of decalcification. This study was taken up by Chappet and Mourisquand on the cords of subjects having presented the clinical signs of nervous osteomalacia and in the three cases examined they found constant lesions characterized by black blocks scattered throughout the different columns. Therefore the lesions are not systematized; this degeneration was recent in all three cases and could not have dated back more than about a month, this being the time when the nervous symptoms developed.

These observers—without wishing to come to absolute conclusions—believe that “this change sufficiently explains the incomplete paraplegia presented by the patients, as well as the always marked exaggeration of their reflexes.” Hence from the viewpoint of the osteomalacia *per se* nothing can be gleaned.

In most cases the muscles become involved early in the process. There is notable atrophy accompanied by fatty transformation, as in the case I shall report and others, including Tixier’s case. Freidrich supposed that the process in the muscles was of inflammatory origin.

Examination on the peripheral nerves has given variable results and in his personal cases Meslaye was unable to find any pathologic change whatsoever.

As to the kidneys, a manifest congestion has been noted in some instances, with dilatation of the pelvis and ureters. Vesical calculi have also been found, having as a basis calcium phosphate and magnesium carbonate.

All the changes arising in the urinary system may finally end in complications terminating in death as is illustrated by Davis-Colley’s case.

The age of the child is not stated or the date of onset of the disease, but it is said that the patient was treated for ten years for

fracture of the femur. The patient was pale and sickly looking and early in life had a waddling gait, pes valgus and genu varum.

Later on there was a fracture of both arms. The long bones, especially the tibias, were soft and flexible. The skull presented craniotabes. After a time the lower jaw became thickened at the angles. The swelling at first soft, became hard later on. There was a tricalcic phosphate deposit in the urine and before death there was some pus.

Eight months before death phosphatic calculi were detected, death being due to a pyelitis the result of the calculi. The ureter and right kidney were filled with calculi.

The patient's head was broad, the forehead deformed and the upper limbs twisted. The thorax was not rachitic in type. The usual medication—iodine, iron, mercury—had no effect whatsoever.

Autopsy verified the diagnosis of osteomalacia.

#### PATHOGENESIS

The innumerable number of explanations offered in respect to the pathogenesis of osteomalacia is enough to show that there is none that can apply to all cases of the affection. I shall merely give a summary of the most important in order to give a general idea of the subject as it now stands.

Bouchard, in his work on diseases due to defective metabolism, places osteomalacia among the acid dyscrasias and says: "Osteomalacia appears to belong to these pathogenic states in which there is an exaggerated formation or an insufficient combustion of lactic acid and also oxalic acid, according to Benke." He also states that the bones in this disease present an acid reaction and abnormally contain lactic acid.

Now, when lactic acid accumulates in the osseous tissue to the extent of producing an acid reaction it creates a condition resulting in the reduction and elimination of the phosphates of calcium. The living organism incessantly manufactures acids, but it also incessantly destroys them in physiological conditions. There are morbid states in which oxidation of the organic acids decreases, or at least these acids accumulate from this influence.

Following Bouchard's statements a number of experiments were

undertaken in respect to the chemical composition of the bone in osteomalacia and in some cases, as Schmidt-Weber has pointed out, lactic acid was found in the bone marrow. But on the other hand, Virchow and Momsen found that the fluids of the bone marrow in some cases of osteomalacia that they examined were alkaline in reaction. Hence it is possible that lactic acid is present during a certain phase in the evolution of the disease and later on disappears.

From the viewpoint of pathogenesis and diagnosis, the presence of lactic acid in the urine is not of much importance, because Langendorf, Momsen and Spira have found it after excessive muscular exercise and Moscatelli confirmed these findings by detecting lactic acid in the urine of soldiers after long marches.

Observers have not been content to merely incriminate lactic acid as the provocative agent of this disease and some have thought that the same results were produced by acetic and carbonic acids. Experiments carried out by Tripier, Gayet and Bonnet lead to this conclusion.

Gelpke believes that osteomalacia is the result of an excessive physiological absorption of bone, as occurs in osteomyelitis, caries, etc.

In his experiments on the metabolism of calcium, Etienne followed the progress of lime from the time of its ingestion in the food and on the other hand pathologic decalcification of bone, up to its elimination or deposit in the viscera, and found that the results obtained were in opposition to what takes place in osteomalacia in adults. In these cases the intact walls of the arteries do not fix  $\text{CaO}$ ; the calcium thrown *en masse* into the blood takes place spontaneously, a fact which shows that absorption of lime salts, especially  $\text{CaCl}_2$  in large doses does not produce calcification of the vessels and that therefore the calcium is eliminated in totality.

In opposition to this Etienne found in his little patient that calcification of the walls of the aorta took place while decalcification of the skeleton was in progress, a fact which goes to show that the mechanism of osteomalacia should not be sought for in a general cause, such as chemical changes of the blood, or any other which acts both upon the aortic walls and bone, because it is, in reality, due to the loss of power of the cell to dissolve calcium, either on account of some histologic change of the osseous type, or by transformation

of osseine into a special type of albumin described by Bence-Jones, or by some other mechanism as yet unknown.

*Infectious Theory.*—Zurn described a micrococcus in the bone marrow of osteomalacia and later on, Petronne thought that he had found the specific agent of the disease in Winogradski's micrococcus. He even stated that he had found it in the blood of these patients and went so far as to assert that he had produced the disease in dogs by infections of pure cultures of this organism.

Close study by other observers soon ruined this theory; among others was Marpurgo, who published the results of his experiments on white rats. Having histologically examined certain viscera of rats dying with typical symptoms of softening of the bones, he noted the presence of diplococci readily stained by analin dyes and Gram-negative.

Having injected a culture of these organisms in forty-four healthy rats, twenty-seven of the animals presented the same symptoms as those from whom the culture was obtained.

But later on, other experiments were undertaken with the result that although the infectious theory of osteomalacia may not have been completely discarded, it has, at least, been profoundly forgotten.

*The Para-infectious Theory.*—After the theory of infection had lived and died, the theory of para-infection appeared upon the scene. Thus when the infections had become better understood and that the idea of a specific microbe for each disease had had its day, it became known that a given germ could cause various affections according to its virulence, the soil offered by the patient and other conditions.

Hence those who upheld the theory of para-infection maintained that osteomalacia should be regarded as a disease, caused not by a given pathogenic germ, but by a distant action of some infectious process on the osseous system. To verify this assertion they undertook careful search into the patient's antecedents both from the viewpoint of symptomatology and pathogenesis.

In a good number of cases, particularly in adults, an infectious state was discovered before the advent of the symptoms of the osteomalacia which, according to these observers, could be the cause of the development and evolution of the disease. For example, Adenot attributed the osteomalacia in his case to a previous puerperal infec-

tion, while Duval's patient developed the disease six months after a typhoid fever and about eleven years after her first pregnancy. A close study of the evolution of the osteomalacia led Duval to consider it as an infectious process, as it was accompanied by pyrexia, it evolved in outbursts, occurring after more or less distant intervals.

Bouley and Hanot remarked that osteomalacia is not only accompanied by pyrexia, but by suppuration as well and Adinot maintained that infections certainly play a very important part in the onset and evolution of the affection, especially of osteomalacia of pregnancy; that castration probably does not act in a direct or reflex way upon the process, as Fehling maintained, but simply by preventing pregnancy which favors the evolution of the morbid process, just as it influences other pathologic states.

Arcangeli, of Rome, considers osteomalacia, rickets and Paget's disease of the bones as three forms of the same infection caused by a diplococcus in the bones. According to this observer the organism resembles the staphylococcus albus, but its specific characters are made evident by a special liquefaction of gelatin, the reproduction of the disease in rats, guinea-pig and rabbit, the manner of agglutination, etc. All three are contagious diseases, because there are houses in which cases of rickets and osteomalacia occur.

Wright's vaccine, he maintains, gives good results in these affections and the same vaccine can ameliorate the three forms of specific osteolytic diplococcia.

Potier is very reserved in his opinion in respect to the above microbic theory and very properly recalls the work of the French school which is opposed to it. The notion of the intimate relationship uniting the functional perturbations of the vascular glands and the evolutive changes of the skeleton is of rather recent date. In the case I shall report I shall refer to a certain number of symptoms of suprarenal insufficiency in the little patient, such as high blood-pressure, cutaneous pigmentation, Sergent's white line, etc.

Given the bonds which appear to unite osteomalacia with the functions of the suprarenals there seems to me to be an element here that should be taken into consideration in the therapeutic indications of this curious morbid process. The arrest of development due to thyroid insufficiency (myxœdema) is no longer questioned; acrome-

galia, gigantism, adipose-genital degeneration and skeletal changes seem to be related to changes arising in the hypophysis. Hutinel compares the special dystrophy in adolescents described by him to cases of pituitary dwarfism reported by Burnier.

In 1907, Bossi advised the administration of *adrenalin* in cases of osteomalacia. Various experiments showed the marked influence of this product on the fixation and retention of salts entering into the composition of bone. And in reality, since this time a certain number of successful results have been reported from the use of this drug. Nevertheless, although the functional insufficiency of the suprarenals in certain cases of osteomalacia is undeniable no mention is usually made of the classic signs of defective functions of these glands in most cases of this disease.

On the other hand, in Tixier's case I have been able to discover a certain number of signs which would seem to plead in favor of exhaustion of these glands, because, besides asthemia, the blood-pressure was low, there was unusual pigmentation in certain parts of the body and lastly, Sergent's white line was distinctly produced which, for many writers, is a good diagnostic sign of functional insufficiency of the suprarenals.

In all cases reported of acromegalia, gigantism and adipose-genital degeneration described by Frohlich, Bartels and Launois as dystrophies, it seems more likely that they are related to changes in the hypophysis, especially to hyperfunctioning of the anterior lobe of the gland.

May not there be reason to suppose that the peculiar condition known by the name of tardy rickets with obesity, dwarfism and delay in the development of the genital apparatus result from hyperfunctioning of the posterior lobe of the same gland?

It would therefore seem that the hypophysis may make giants or dwarfs according to whether its functions are increased or diminished. It is possible that a change arising in this gland, a hyperfunctioning of its anterior lobe for example, may be incriminated in cases of osteomalacia which are accompanied by many symptoms similar to those of tardy rickets, muscular impotence and obesity, but in this respect one must be reserved in one's opinion.

Thus we come to the ~~on~~ his theory which has and still holds an

important place among all the explanations offered for the pathogenesis of this affection, especially of osteomalacia in adults. The ovary, by an increased or diminished secretion, is supposed to be the cause of the bone disturbances of osteomalacia. Curattullo and Tarulli castrated bitches and noted a decrease of the elimination of phosphates and then, by injecting ovarian extract, they found that the phosphates were eliminated in larger amounts.

Gayet and Bonnet experimentally verified these findings but stated that this fact would not allow one to conclude that there was a bond between a pathogenic action of ovarian disturbances and osteomalacia.

In France, Fochier was the first to perform castration following a *cæsarean* section on a patient with osteomalacia; the result was successful. Some years later, Fehling performed bilateral castration in a case of osteomalacia; the outcome was successful and he then advised this operation as a treatment of this disease.

A number of castrations were done in different countries, but the results were far from attaining the 80 per cent. success as given by Fehling, and this might be expected because the affection is met with in boys as well as in adult males, while since osteomalacia occurs in children of both sexes, the question of the internal secretion of the sexual glands cannot be taken into consideration because of the rudimentary development of these glands. Therefore I believe that this theory, quite as much as all the others, may be accepted in a certain number of cases, but that it is far from comprising all, even in adult females.

Finally, a word may be said in respect to Poncet and Leriche's theory which envisages osteomalacia as the result of a disease of the bone marrow, considering that this disease, no matter what may be its form or date of onset, is a syndrome presenting a uniform clinical aspect, although they recognize that it possesses a very varied etiology and many mechanisms. For instance they say that there are cases of osteomalacia of ovarian, suprarenal, thyroid origin, etc., due to insufficiency or secretory changes of these glands. But these glandular disturbances are secondary causes, but what is important is the primary causal factor of these pathologic states. The cases published by these observers, as well as their experimental work, lead Poncet

and Leriche to assume that tuberculosis—in that it is an infectious disease—is often the primary etiological factor.

#### SYMPTOMATOLOGY

*Onset.*—This varies, but is generally slow and silent, so that when the diagnosis of osteomalacia is to be made, the disease has already existed for some time. Hence the impossibility of making a sure diagnosis of the disease at its onset. The two essentially characteristic symptoms noted in children are pain and genu valgum.

*Pain.*—This may be said to be the most constant symptom, usually the first to appear, as is clear when one reads the reported cases; but on the other hand, there are instances in which pain appears late in the evolution of the morbid process at a time when the disease is fully developed.

In females who have been pregnant the pain is localized in the pelvis, in the adult male in the spine and in children in the limbs. The pain is spontaneous but is accentuated by pressure or movement, so that these patients avoid all motion. It varies in intensity, being sometimes fleeting or erratic, but is more prone to be fixed and always localized in the same spots. The pain recalls the osteocopic pain of syphilis, while its frequent seat in the joints at the onset of the affection has often resulted in the erroneous diagnosis of rheumatism.

Trousseau and Lassègue described a peculiar mental state in these patients, characterized by hyperæsthesia which may attain such an extreme degree that they gave it the name of *nervous susceptibility*. Thus Lobstein described this nervous state in his case, in which the mere approach of a fine linen handkerchief was enough to provoke violent pain in the limbs.

But this susceptibility is rather more a kind of automatic reflex produced by the muscles which contract in order to immobilize the limbs in order to prevent still severer pain resulting from movement.

The second symptom, genu valgum, is often the first to attract attention and is often the only one noted at first in children. Therefore it is of great importance and should always be looked for. It is frequently bilateral and continues its evolution regardless of treatment.

*Contractures.*—These ensue usually in an advanced stage of the

process. They may become permanent and are above all seated in the adductor muscles. The knees become so tightly pressed against each other that the hand can hardly be passed between them. In one of Meslaye's patients the permanent contracture maintained flexure of the legs on the thighs and absolutely pressed against each other.

In one of my cases contracture of the flexor muscles existed to such a degree that it gave rise to very marked pes equinus and it is important to remark that this contracture developed progressively and remained permanent.

Latzko states that in his cases, the contracture continued during narcosis. Finally, it is to be remarked that it is contracture of the dorsal muscles which plays the essential part, primordial so to speak, in all the deformities of the spine encountered in osteomalacia.

*Disturbances of Motility.*—Muscular weakness will be noted variable from one case to another and according to the phase of the evolution of the affection. This weakness may reach an extreme degree, the subject being unable to raise the feet off the bed and in order to effect this movement he is obliged to support the thighs. A point interesting to remark is that these disturbances of motility generally begin in the lower limbs and rapidly progress attaining a high degree, and then appear in the upper limbs. The weakness may reach such a point in the upper limbs that the patient may not be able to use them. For both Koppen and Renz this muscular weakness has a great importance; the latter observer has made a very good study of it and he especially insists on the myasthenic character of osteomalacia. As to the explanation of this paresis, Renz supposes that it is due to a transversal lesion of the cord resulting from the exudate from osteomalacic peripactimeningitis.

*Gait.*—This is peculiar in this affection, being slow and hesitating like a duck, this being due to the fact that the patient cannot lift the legs, so that in order to walk the subject is obliged to bring the leg forward by raising the hip-joint and at the same time throwing the trunk to the opposite side. Latzko says that as an essential cause of this peculiar gait a bilateral dislocation of the hip is present having taken place during the evolution of the osteomalacia.

In respect to gait I would draw attention to a very special phe-

nomenon observed in these patients when, while lying on the back, they are requested to turn over on the belly. One then observes a complexus of movements especially effected by the trunk, aided very little by the upper limbs, particularly in bending them and leaning on the elbow of the side opposite to the direction that is to be taken; and then by an effort rather of a start than of muscular contraction *reptation movements* are seen to take place, thanks to which the patient attains his end after very long and difficult effort.

These reptile-like movements are like those observed in reptiles when they wish to turn from one side to another, taking place principally in the muscles of the body with little help from the feet, especially the anterior.

The state of the reflexes varies from one case to another. Usually they are normal but there are instances in which they are increased or diminished, while some may be wanting. These differences may perhaps find their explanation by the phase of the evolution of the process at the time the patient is examined and in particular the pathologic state of the cord. In many instances the cord being indirectly compressed by curvature of the spine or directly compressed by a peripachimeningitis or by lesions of the columns of the cord, may explain the difference of opinion of observers in respect to the reflexes.

*Electrical Reactions.*—In several of his cases Koppen noted a decrease of the galvanic and faradic sensibility, especially in the leg the most involved. He also noted tremor of the limbs and tongue. In half of the cases examined by Velits he found a rhythmical tremor of the lower limbs during movement. In one of his cases, Meslaye observed this infirmity combined with muscular weakness at the onset of the disease and states that the former phenomenon was the cause obliging the patient to take to the use of crutches.

*Urine.*—This is cloudy and whitish, giving rise to a deposit resembling curdled milk, and is present in large amount according to Collineau. Stansky states that in a case in which an analysis was made by Burrue, it showed an excess of calcium phosphate and a large amount of gelatine. In one of Kilian's cases sodium phosphate was found in excess.

In three of Collineau's patients there was an excess of lime phos-

phates and the same fact has been noted by Litzmann. And nevertheless, regardless of the considerable number of recorded cases this subject is still discussed. Thus Schutzemberger found less phosphates in the urine of an osteomalacic patient than in normal urine; Langendorff and Momsen likewise found a decrease of phosphates. Therefore according to these observers one would be inclined to suppose that in many cases the amount of phosphates eliminated in the urine in osteomalacia may be less than normal.

Fehling carefully examined some cases of osteomalacia and came to the conclusion that probably during the onset of the disease the amount of phosphates eliminated in the urine is larger than normal, but as the affection progresses the quantity decreases and finally attains an abnormally small percentage.

As to the question of the presence of lactic acid in the urine it is still more moot than that of the phosphates.

Collineau was the first, I believe, to call attention to the abundance, fetidity and acridity of the sweat, which may be so intense as to cause pruritus.

*The Blood.*—The hematology of osteomalacia has given rise to much study. Von Jaksch showed that in a goodly number of cases the alkalinity was greatly diminished and attributed this to the formation of organic acids. But on the other hand, Fehling noted that this diminution was not constant and that it cannot be given the pathognomonic value that some are inclined to attribute to it. As to the morphology of the blood in this disease space forbids entering into farther details.

*Genital Functions.*—In spite of the numerous cases reported from various sources the results obtained by ovarian castration leave much to be desired. Various observers have found the menses irregular, prolonged or with metrorrhagia after the menopause. Others have noted normal menstruation in their patients.

On account of such different findings I shall abstain from any firm conclusion in reference to the influence of the disease on menstruation, but I would emit the very plausible hypothesis that the disease itself has little influence but that the etiological factor or factors which provoke or accentuate the morbid process are the cause, and, on the other hand as these factors are probably quite

numerous it may be that the very number may explain the differences observed in the menstruation of these patients.

Fehling was perfectly correct when he stated that osteomalacia is evidently related to sexual life and that from the viewpoint of prophylaxis marriage and pregnancy, especially, are to be avoided, but he adds that unfortunately, this is difficult to obtain in the social midst in which this disease occurs.

I will here give a detailed case report of osteomalacia in a girl thirteen years of age and I take this opportunity to thank Professor Hutinel for permission to publish it.

CASE.—Female, *æt.* 13 years, entered hospital because she could not stand on her feet. She measured hardly one metre, eleven centimetres in height and looked more like a child of six years.

The mother appeared to be in good health. There are three other children older than the patient who are said to be in excellent health. The father, an alcoholic, died accidentally.

The patient was born before term, at eight and a half months, was small, weighed five pounds and only measured 48 centimetres. Breast fed until the age of eleven months at which time she was sent to the country at her grandparents. First teeth at eight months, began to walk at fourteen months and to talk at ten months; the development appears to have been normal.

Up to the age of eight years there was no illness; patient walked well and no deformity of the limbs was evident, but she was small, thin and at the age of five only weighed 13 kilograms. However, at about the age of three the patient was ill for about a fortnight with headache, vomiting and pyrexia and at the time the attending physician had spoken of meningitis.

At eight and a half years began to complain of pain in the left knee and from this time on the disturbances of the nutrition noted developed in the following order:

The pain in the knee was treated in the hospital where a genu valgum was discovered. This was reduced and the limb put up in plaster. For five months she remained immobilized and during this time she began to grow large, the abdomen assuming an exaggerated development. The patient had always been thirsty and urinated large amounts. The mother states that the urine had always been

clear and frothy, but apparently sugar and albumin had not been searched for. There was no œdema at this time, only enlargement of the abdomen.

The child was sent to Berck (a seaside resort) and there remained for three months. When she returned she walked much better and the abdomen was smaller. From nine to eleven years her health was fairly good but obesity was excessive, the weight reaching 30 kilograms. Thirst was marked and the child drank continually; the urine was clear and passed in large quantity.

In May, 1910, the patient was thirteen and was suddenly seized by generalized œdema. Admitted to hospital she remained five months under treatment. At this time fourteen grammes of albumin per litre were found in the urine. She was discharged in September greatly improved, she could walk easily and only weighed 27 kilograms.

In June, 1911, the patient began to tire easily while walking and soon was incapable of standing and it was in this condition that the child came under observation. The small height and obesity of the patient was striking, quite as much as the deformity of her limbs and weakness. The fat was especially marked over the abdomen and thighs. The complexion was pink and eliminated any idea of cachexia. There was not a trace of œdema.

The lower limbs were manifestly deformed. Very marked genu valgum on the left, and a deviation such that at first this deformity was suspected of being the principal cause of the impotency. Incurvation of the femurs, especially on the right. Distinct tumefaction of the epiphysis of the knees and tibio-tarsal joints, abnormal laxity of the ligaments which allowed extensive lateral movements at the knees. The thighs were large but soft and when seized in the hand thin muscles without energy could be felt under the mass of fat; the adiposis and gracility of the limbs was still more distinct when the legs were palpated.

The feet were in marked equinus but this position, which was easily corrected, was especially due to the weakness of the extensor muscles. The toenails were striated transversally and strongly incurved; no cyanosis, no cutaneous lesion other than that the skin was dry and somewhat scaly.

The abdominal wall was laden with fat, but the liver and spleen were not hypertrophied. No ascites, no venous ectasis.

As in rickets, the thorax was flattened laterally and seemed too narrow for the abdomen.

The upper limbs were thin and without strength; the weak muscles were surrounded by a thick covering of fat. Swelling of the radial epiphyses at the wrist. No pulmonary or cardiac lesions, no hypertrophy of the heart or disturbance of the rhythm.

The head was not very large but the frontal and parietal bosses were projecting; the nose was flattened at its root; teeth in good condition; nasal breathing sufficient. No headache, no ocular disturbances, pupils equal, patellar reflexes a little sharp, no clonus, no Babinski.

Severe grade nephritis, urine clear, total amount in 24 hours 1100 c.c. to 2000 c.c.; albumin 50 centigrams per 1000 c.c., no sugar. Blood-pressure was normal.

The child was put on a diet without salt from August 9 to 20 without any effect upon the body weight.

During the following weeks the patient presented attacks of pyrexia from time to time, in temperature reaching 101°, 102° and even 105° F. without any other appreciable symptom, the attacks lasting three or four days. It should be noted that the intradermic reaction done at the time the child was admitted to hospital was positive and that the polyuria was in no way changed during the febrile attacks.

In October the thyroid treatment was begun without any change in the body weight or diuresis, Wassermann negative.

By radiography the cartilages of the epiphyses were found to be considerably broadened, divided into two zones, a pale one in the epiphysis—chondroid zone—and a darker one—chondro-calcic—in the diaphysis and distinctly separated from the neighboring bone. The epiphyses were increased in size, there was incurvation of the diaphyses, the central canal was enlarged and in places in the long compact bones or in the ossified epiphyses could be seen paler areas unquestionably corresponding to zones of decalcification.

How long these lesions had been present it is difficult to say. Up to the age of eight years the child walked and developed normally,

but there is nothing to prove that they were perfectly normal before. Walking and standing were now no longer possible and if the patient was asked to raise the foot from the bed the act was accomplished with difficulty and the slightest touch was sufficient to overcome the effort. When the child turned over in bed she did so by a slow movement of reptation, frequently ineffective. To sit up was very difficult. The movements of the arms appeared to be easier and those of the hands were not too awkward, but the child could not grasp the dynamometer when the instrument was placed in the hand and was incapable of making the needle move. The muscles of the neck and face were less weak; she can whistle and turn the head without any trouble.

Electrical examination showed a very marked faradic and galvanic hypo-excitability, especially on the left.

The alimentary glycosuria test was positive. The child was given 100 c.c. of glucose syrup and the urine contained sugar four hours later which only disappeared at the end of ten hours.

Albumin was constant, usually about 50 centigrammes per 1000 c.c. The child was much stronger in 1910, when she had a serious nephritis. The albuminuria was not orthostatic because the child was always lying in bed; neither was it a digestive albuminuria. Briefly, it was a persistent albuminuria due to renal lesions.

November 11, 1911.—Methylene blue (1 c.c.) was injected subcutaneously, but the urine did not become colored. Five hours after the injection chromogen was found and required exactly ten hours for elimination. Therefore the methylene blue passed into the urine with difficulty.

November 13-15.—Eight grammes of salt were given daily and nevertheless the amount of urine did not decrease, and increase of body weight was hardly appreciable. Hence chloride retention was absent.

November 16-18.—Twenty grammes of urea were given *per os*. At first a slight diuresis ensued, but the elimination of urea was late and incomplete. There was consequently a mild but appreciable uric retention. The specific gravity of the urine was 1004; the amount of urea did not reach five grammes in twenty-four hours and the coefficient of demineralization was quite high, being 12.57.

A striking feature of the case was the short stature and excessive obesity of the patient. But in spite of the nephritis there was no trace of œdema or myxœdematous infiltration. The hair was silky, the intelligence normal, the fontanel had long since closed and there was no disturbance of the circulation or sensibility. The movements were slow, but the expression was normal and there was no evidence of delayed mental development.

The thyroid appeared to be normally developed, and there were no symptoms that pointed to any functional disturbance of the gland. In fact, thyroid treatment, continued three weeks, gave no result.

Several radiographs of the sella turcica showed that its dimensions were normal. Examination of the eyes showed no change and especially there was no temporal hemianopsia.

The patient was discharged on May 26, 1912, having at this time 50 centigrammes of albumin per 1000 c.c. She continued well until September 4, 1912, when she menstruated for the first time. The menses were very profuse and lasted ten days.

October 2, 1912.—Menses recurred. For the past month anorexia. The patient slept well, but the mother thought that the child was feverish. Nocturnal temperature normal. A little œdema of the legs towards evening.

Two years later the patient was admitted to hospital. The obesity and dystrophy were more marked while the osteo-muscular atrophy daily increased. The menses, at first regular, decreased in amount until they no longer occurred for three months but afterward they were irregular and then completely disappeared after March, 1914.

May 4, 1914.—The patient was thoroughly worn out, only the eyes retained their normal brightness. The child progressively emaciated, menstruation had ceased since March last, the hair on the genitalia was falling out, likewise on the scalp. Movement was painful, the arms could not be raised and nevertheless the joints were not painful. Excessive pain when the limbs are palpated, especially the lower limbs and over the epiphyses.

Very marked deformity of all the bones; scoliosis tremendous. On account of the deformed pelvis the abdomen projects. The sluggish intestines filled with feces could be easily palpated.

The patient, who weighed 27 kilogrammes three and a half years previously when she first entered hospital, now weighs, at the age of sixteen, only 15 kilogrammes, 400 grammes.

Auscultation of the lungs revealed little other than that the right apex was somewhat suspicious.

When the child was first admitted there were 50 centigrammes of albumin and a marked glycosuria; at present the sugar has disappeared and the amount of albumin is about the same. Blood-pressure was low. Pulse 120, very weak, temperature normal.

Three years ago the height was 1 metre, 10 centimetres; at present it is 92 centimetres.

*Autopsy* (November 26, 1914).—Bilateral atelectasis. Heart normal. Liver large, spleen small. Suprarenals appear macroscopically normal. Kidneys very small and white, each weighing two grammes. The skull is soft and depressible; pronounced incurvation of the diaphyses and a pseudoarthrosis of the left femur. All the bones are soft and easily fractured. Ovaries and thyroid macroscopically normal. Brain presents no morbid change.

*Temperature*.—In spite of all the theories of infection and para-infection invoked for explaining the pathogenesis of this affection, as well as the cases published in support of these theories, I believe that, as a general rule, osteomalacia is an apyretic process and that if the temperature does go up during its evolution, the cause must be looked for in some intercurrent affection, pulmonary or other, rather than in the disease itself.

The same may be said in respect to the appetite, which is usually good until the ultimate phase of the affection is reached when, in fact, all the vital phenomena are likewise seriously compromised.

*Decrease of the Stature*.—Collineau noted a decrease of the height in twelve cases. Frank's patient reached the size of a four-year-old child, while Dronineau and Meslaye observed the same phenomenon in their patients.

This decrease of the stature is not always a premonitory symptom of the disease as it may occur late in the evolution. It may also be due to fractures, deformity of the skeleton, sinking of the spine and especially to deformities of the lower limbs.

*Deformities of the Skull and Face*.—The bones of the head are

involved late in the progress of osteomalacia so that the skull is rarely deformed. But in Davis-Colley's case the lower jaw was deformed, giving a peculiar aspect to the face, while in J. Voisin's patient a curious deformity of the skull ensued, especially marked in the frontal region.

The bones of the pelvis in osteomyelitis are described in all text-books on obstetrics, hence little need be said, other than that the promontory encroaches upon the pelvic cavity, while the pubic branches become parallel to each other, forming the classic "bird's beak."

The two iliac bones soften and offer multiple deformities.

*Pseudoarthrosis*.—The non-union of fractures need not of necessity occur in osteomalacia as perfect consolidation is known to take place in these patients, even with multiple fractures which may unite very quickly. But even when union ensues, the callus does not remain because a process of destruction invades the bones and the callus, all becoming soft, so that a pseudoarthrosis ensues at the point of fracture. Saint-Gilles even concludes that this is the final outcome of all fractures in osteomalacia, but Baake maintains that he has observed permanent union.

The softness of the bones may reach such a degree that they can be bent like a green stick or given any shape desired, a fact which explains the occasionally extraordinary shape of the limbs encountered. They can even be twisted into the shape of a drill.

Briefly, the deformity and decrease of the stature which occur may become arrested, the fractures seem to consolidate and from the ensemble of these conditions results the characteristic appearance of the limbs, trunk, and thorax. It would be a matter of difficulty to describe all the changes met with, but the osseous changes in the spine and pelvis explain the decreased height of the subject.

The deformities are sometimes only partial, in which case the pelvis is the seat in adults, the limbs and thorax in children. In quite a number of cases it was the decrease in the patient's stature occurring at the onset of the disease that led to a diagnosis of the process.

Albumin in small amount is sometimes found in the urine in osteomalacia. Much attention has been paid to an albuminoid body

—also called albuminodentoxidrate or hemialbuminose—referred to by Macintire and Bence-Jones, and also found by Kuhn, Langendorff and Momsen. It has been regarded as a nitrogenous compound by some, similar to albumin, but not identical to it. It would offer a real interest if it could be regarded as a pathognomonic product because Virchow found hemialbuminose in the bone marrow in osteomalacia, but since then Fischer has found it in normal bone marrow. This body has been likewise found in diseases other than osteomalacia. Thus Lasson detected it in experimental nephritis, and Kuhn, Schmidt, Muhlheim and Salkowsky have shown that hemialbuminose is produced during digestion.

#### THE CLINICAL FORMS OF OSTEOMALACIA

Concerning the forms of osteomalacia, I shall not describe those occurring in adults, or those encountered in elderly subjects and known by the term of the senile form of osteomalacia. The following remarks are confined to osteomalacia in childhood, or *juvenile osteomalacia*.

Very frequently mistaken for rickets, one must be very reserved in respect to intra-uterine osteomalacia described by the older writers, as well as osteomalacia said by Stansky to have been met with in the newly born. Rehn was the first to describe juvenile osteomalacia as a morbid entity and after this Davis-Colley reported a case of infantile osteomalacia. Herman (1888) studied a patient presenting deformities of the limbs but he believed that it, and the cases described by Rehn, were instances of rickets.

Herman supposed that if it were admitted that osteomalacia was due to absorption of the calcium salts of the bones it would be very difficult to understand how absorption could act on bones hardly calcified. Hence it is at a more advanced age, when growth of the skeleton has reached an almost normal degree that softening of the bones is met with in children as well as in adults. But Meslaye, in the case of a thirteen-year-old child, found all the clinical signs, and the diagnosis was further confirmed by a microscopic examination of the bones and he says: "From the viewpoint of histology this case is similar to rickets on account of the irregularity of ossification that was noted in the head of the humerus, as well as the presence of

osteoid tissue between the subsisting lamellæ, but it differs from it by the very nature of these lamellæ. The presence of osteoblasts, and especially myeloplaxes upon which the principal part of the bone absorption falls, shows that anatomically this case should be regarded as a variety of generalized chronic osteitis, with progressive rarefaction of the osseous substance and fibrous transformation of the intermediary medullary spaces."

True osteomalacia—a general disease—is not to be confounded with local osteomalacia. It is to the latter variety that Larayenne's case belongs that he has described under the name of traumatic osteomalacia. In this case the affection was local, as is observed after infection or nervous affections. In local osteomalacia of traumatic origin nervous lesions usually exist and an examination of the nerves will reveal neurotic disturbances.

Quite frequently one meets with, and Destot has called attention to this fact, disturbances of decalcification with sharp pain, following severe traumata or fractures.

From the anatomical standpoint, such a softening of the bone, which has no tendency to become general, has exactly the same microscopical aspect as the generalized form of osteomalacia. In the present state of our knowledge it cannot be said that the classification of osteomalacias reposes upon a very solid basis, and for this very reason, in examining the various recorded cases of so-called *essential* osteomalacia, one is struck by the fact that many differences exist among them—age, determining factors, pathology and probably pathogenesis—all offering considerable variations. The process may evolve in an acute or chronic course and invade all or only part of the skeleton, so that we have a series of transitions between local and general osteomalacia.

Necrosis of the bone and osteitis can be readily produced experimentally, but the process of softening has never been created. All attempts made by Gayet and Bonnet to experimentally produce osteomalacia, such as division of nerves, vascular ligature, irritating injections into the medullary canal, etc., have failed.

*Local Osteomalacia.*—This form is more interesting for the surgeon, because trauma is occasionally the origin of the process. Ususally the injury produces localized softening at the point of or

near the seat of the traumatic insult. It is precisely in this group that traumatic spondylitis, or Kummel's traumatic disease of the spine, should be placed.

The intensity of the injury is very variable, often slight, and is generally the result of indirect violence, such as sprain. The age of the subject likewise varies greatly, but many cases occur in females usually obese and from forty-five to sixty years of age. Here is a good illustration of the local form of osteomalacia in an adolescent, due to Gayet and Bonnet.

Female, *æt.* 18 years. Six brothers and sisters died in early life, probably from meningitis. Patient is the only surviving child and has always been delicate.

At the age of nine years she slipped, sprained the foot but continued to walk in spite of the pain.

At thirteen she fell, remained a year without walking, during which time she kept the knee bent and the foot in extension. Then she walked for a year, but both walking and standing caused the patient to tire quickly.

At the end of a year painful contracture of the thigh muscles led to the suspicion of coxalgia, so the joint was put up in a plaster cast, but this was removed in two months on account of pain.

The patient then was admitted to another hospital where successively a diagnosis of coxalgia, then of myelitis was made and finally no precise diagnosis was reached. As the knee had a tendency to become flexed another plaster cast was applied, but this also had to be removed in three months on account of severe pain and swelling of the foot.

Finally she was admitted to Professor Ollier's service where a radiographic examination revealed a lesion at the lower end of the tibia; there was an old juxta-epiphysary fracture at this end, the fragment of the epiphysis had slipped forward bringing the foot along with it. The bones of the tarsus were seriously deformed and difficult to recognize. All the bones of the foot presented a remarkable transparency.

Given these lesions an operation was imperative for straightening the foot and to reduce as far as possible the deformity resulting from

a union in bad position. It appeared as if this old fracture was the origin of all the disturbances presented by the patient.

Astragalectomy was done, the operation being difficult on account of the softness of the bone, but this facilitated the second step of the interference, namely, the removal of that part of the tibia which projected, as the bone could be readily cut with the knife.

The softness of the astragalus was such that by simple pressure it could be flattened out in any direction like soft rubber.

The results of the operation were at first satisfactory, the wound healing by first intention and at the end of a few weeks the patient was sent home on account of some pulmonary symptoms which, it was feared, were those of the onset of tuberculosis.

The pain in the limb subsided but the pulmonary lesions rapidly progressed.

The prognosis of these bone lesions, leaving aside surgical interference or when this is not possible as for example in spondylitis, is rather dark in that they have no tendency to spontaneous recovery. The pain is persistent, obliging the patient to remain in bed or at least confining him to the room.

On the other hand, surgical interference gives happy results when the lesions are accessible; it has a powerful action over the pain, allows the bones to be straightened and recovery can thus be obtained. The operation consists of trepanation followed by removal of the diseased structures, simple osteotomy, for correcting the deformity, cicatrization and consolidation taking place normally.

Finally, I would refer to Kilian's division of the disease into osteomalacia with softening and osteomalacia with fragility of the bones, as well as Kuhn's point of view, who makes a distinction between the forms of osteomalacia based upon etiological conditions, hence admitting a syphilitic, tuberculous, scorbutic, scrofulous osteomalacia, etc.

#### DIAGNOSIS

As I attempted to show when speaking of the symptomatology, pain is the chief symptom of osteomalacia. During the painful phase of the process the other symptoms of the disease are not characteristic. Therefore spontaneous or provoked pain in the bones

the intermediary structures remaining indolent, is the first symptom noted.

The pain at first has quite a striking resemblance to that of syphilis, rheumatism or gout. Syphilitic pain—osteoscopic pain of the tertiary phase—is located especially in the epicondyles and tuberosities of the tibia, etc. But in osteomalacia the pain may be in the spine or pelvic bones; a condition never observed in syphilis. The pain of syphilis is especially marked at night—perhaps due to the warmth of the bed—while the pain of osteomalacia subsides with rest in bed and only becomes exasperated by pressure.

It is not uncommon, especially at the commencement of the disease, for the pain of osteomalacia to localize in the joints, so that the diagnosis of rheumatism is made and since there is usually apyrexia, chronic rheumatism will be the most likely diagnosis. But in the latter process the joints are usually swollen and red, while the pain does not remain fixed for any length of time in the same joints. It is more prone to occur in the knees, shoulder, elbow and small joints of the hands and feet.

However, it cannot be said that there are any clearly distinctive characters, because occasionally one may meet with all the signs of pure osteomalacia as has been pointed out by Latzko who noted pain in the knees and tibio-tarsal joints in osteomalacia.

Finally, the pain occasionally is temporary in character in osteomalacia, like the rheumatoid pain disappearing for a certain time and recurring some years later.

The pain of acute gout is very sharp and increased by the very slightest pressure, but it appears in the form of paroxysms and their morning onset, the seat of the pain in the metatarso-phalangeal joint of the thumb, the swelling, redness and venous ectasis of the region involved leaves no doubt as to the true nature of the process.

*Differential Diagnosis with the Neuritides.*—In one of Duval's cases of osteomalacia the patient complained of pain above the sacrum, tolerable while resting, but very sharp when the least movement was made, especially bending forward. There was also pain in the lumbar region.

In one of Koppen's cases the pain was seated along the course of the sciatic and this observer supposes that the nerve was involved

in the process, while Duval, in one case, noted that movements of the left lower limb were very painful but that the maximum was obtained by searching for Lassègue's sign.<sup>1</sup>

Confusion is easy with coxalgia and the best proof of this is the case reported by Gayet and Bonnet who applied a plaster cast for three months supposing they were dealing with this lesion, but as the pain continued the cast was removed and it was then that it was discovered that the case was not one of coxalgia.

Finally, there are patients who complain of painful spots appearing to follow along the course of a nerve, but in all cases the physician should base his diagnosis on the extent of the pain and its generalization and not suspect osteomalacia unless all the symptoms belonging to it are present as they will not be long in developing.

In the phase of deformity and flexibility one should be suspicious of all knock-kneed subjects in whom the inward deviation of the knees is very pronounced and has developed rapidly with pain and causing difficulty in walking out of all proportion to the degree of the deformity (Berger).

Usually in these circumstances the diagnosis will offer less obscurity, but it is difficult to attach a single deformity to its true cause. Usually in the absence of other functional symptoms belonging to osteomalacia, the diagnosis is practically impossible.

As to syringomyelia the deformity of the spine is very pronounced and frequently in the form of extensive scoliosis or cyphosis. These deviations appear to depend upon atrophy and muscular contractures rather than upon osseous deformity, while disturbances of anaesthesia are characteristic of syringomyelia.

Time need not be taken for the diagnosis of Freidreich's disease, an essentially familial affection, involving the muscles and rarely commencing with pain. The bones are uninvolved in this disease.

It is needless to consider affections caused by hypo or hyper-functioning of the endocrine glands as I have already referred to them when speaking of the pathogenesis of osteomalacia. Tixier's hypothesis regarding the suprarenal glands appears to be quite likely, given the results obtained by treatment with adrenalin.

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<sup>1</sup> "Lassègue's sign," extreme sensitiveness due to stretching of the nerve trunks in cases of peripheral neuritis (Stedman).

It is in respect to the differential diagnosis between rickets and osteomalacia that many difficulties arise, because in quite a number of cases alone the evolution of the process will be susceptible of giving precise data. And for that matter, recent researches have shown that a distinct line cannot be drawn between the two morbid processes, an opinion ably sustained by Professor Hutinel, who has recorded a case of a special dystrophy of adolescents whose symptoms appear to be connected with both varieties of dystrophies.

However, according to Tixier, it would seem not impossible to meet with osteomalacia and rickets with sufficiently distinct characters to enable one to make a precise prognosis and resort to the special therapeutic indications of each.

The interest of this distinction is great, because the prognosis of rickets has not the gravity of osteomalacia. All the symptoms of the onset are about the same in each process. Thus Tixier, in the case of his young patient in whom the evolution was that of osteomalacia, noted a long phase during which pain was about the only symptom, and Meslaye has shown how, at this phase, it is difficult, if not impossible, to make a diagnosis.

Functional impotency beginning in the lower limbs and progressively increasing, is another symptom, but it is above all the changes in the skeleton from the clinical, pathological and radiographical viewpoints that are essentially useful in making a distinction between the two morbid processes.

In osteomalacia the changes arising in the skeleton are notably different from those occurring in rickets, since the increasing in the size of the epiphyses is less evident over those of the lower end of the radius and tibia than in the upper epiphyses of the femur and especially the humerus. For that matter, the upper epiphysis of the humerus gives the impression of a tumefaction, of exuberance and unevenness, absolutely unusual in rickets.

It might be objected that softening of the bones is not manifest, but both Hutinel and Tixier have shown that this symptom is less common in children than in adults.

Radiographs of the bones of the upper limbs in Tixier's case show very different lesions from those encountered in rickets. In the first place, the diaphyses appear to be at least quite as diseased as the

epiphyses; then the end of the diaphysis does not present the cup with its concavity facing the nucleus of ossification in the epiphysis, a condition quite special to rickets.

Finally, independently of the curving of the diaphyses which was very marked, there was transparency of the bone, a manifest inequality in the way in which different parts of the bone retained the rays, giving the appearance of an irregular Mosaic to the radiograph.

Lastly, I would say a few words as to the diagnosis of tardy rickets. According to Marfan, "the essential deformities of growth may be, and in reality are, most usually the manifestations of prolonged or reviviscent rickets." But to reach this conclusion Marfan refers to all the cases he has been able to follow which presented all the symptoms of rickets in early childhood.

A. Polosson likewise expresses himself as follows as regards the relationship existing between tardy rickets and that of early childhood. There is "persistency in the repaired osseous tissue, especially in the epiphyses, of Broca's cartilaginous nodules, medullization of these nodules at the time of puberty and secondary formation of spongoid lesions ending in a deformity of the bone."

The idea of reviviscence of tardy rickets is quite possible, but one might also add that the bone changes appearing at puberty might be provoked by an infection or chronic intoxication of recent or old date, and that in many cases the causative factor of rickets of early childhood may not have become extinguished and hence in reality be the cause of the osseous change—syphilis, chronic tuberculosis, etc.

The onset of the manifestations of rickets is marked by lassitude, especially when the patient arises, a tendency to rest, intellectual torpor, muscular soreness and more or less marked dyspeptic disturbances.

Now, it is exactly *à propos* of this question that I referred to Tixier's case because it gave rise to many contrary opinions from the members of the Society of Pediatrics of Paris in 1912. In point of fact, if this case is attentively examined, one may well ask the reason of the contradictory opinions put forth, especially those of Comby and Marfan, who stated that in these particular instances the manifestations were rather those of late rickets than of osteomalacia. Do not these two clinicians believe that all cases of infantile osteo-

malacia are what they call late rickets or else in this case do they assume that the manifestations are those of late rickets?

With both Tixier—who presented the case—and Hutinel and having myself examined the patient in question, there is no doubt in my mind that the case was one of infantile osteomalacia.

#### TREATMENT

The rational therapeutic indications at the onset of osteomalacia are tonic medications. In accord with most observers, one must favor and reëstablish the nutrition of the osseous system. This is a most natural conclusion, but one difficult to fulfil. The phosphates and tonic medication usually fail. It is easy to advise the patient to live in a dry, warm climate and not to marry. Exercise and hydropathy may be recommended if the patient has not reached the stage where rest is essential, as well as control of the pain and sudden deformity.

But all things considered, these measures are palliative, immobility itself is a cause of denutrition which singularly weakens the action of medicines and other than the phosphates, cinchona, iron, some digestive stimulants, cod-liver oil and arsenic, there is no special indication excepting hygiene.

Among the various treatments advised for osteomalacia there are some that perhaps have given encouraging results. Puerperal osteomalacia has now a somewhat better prognosis, the result of interferences based upon pathogenic theories, but from the more recent results and according to some observers one is led to suspect that the improvement derived from castration has been exaggerated.

And this brings us to another surgical procedure particularly frequent in cases of infantile osteomalacia. This treatment is not directed against the disease itself but deals with the results. As I have pointed out many cases of osteomalacia commence by a deformity of the lower limbs, especially by genu valgum, which is generally bilateral and more marked on one side than the other. An operation is reasonable enough in cases in which this is the only symptom of the disease because the surgeon does not suspect its true nature and he therefore applies a plaster cast or does an osteotomy, and it is only after the operation and immobilization that the little patient at the

time the cast is removed—oftentimes on account of pain—that the surgeon to his great stupefaction finds that not only union has not taken place but that more accentuated dystrophies have arisen and that a series of symptoms are present which were absent before the operation.

It is precisely *à propos* of these interferences that I wish to draw the attention of surgeons, because it seems to me that operative interference increase the rapidity of the evolution of the disease, likewise the immobility to which the patient must submit. To be sure I cannot elucidate the entire question on account of the very numerous pathogenic theories invoked for explaining this morbid process, but what is certain is that after operations the evolution of the affection assumes an acute progress.

For this reason I maintain that before interfering surgically it will be prudent to examine the patient's antecedents with the utmost care, likewise their actual physical condition and their collateral antecedents. In all these cases one will do well to recall Berger's words already quoted, as well as those uttered by Broca: "Be suspicious of genu valgum with a rapid evolution, beginning too late in life for rickets and too soon to be a genu valgum of adolescence, and in which the deformity becomes very severe and continues to become aggravated."

In a weak child, who is becoming slowly cachetic, presenting osseous disturbances especially characterized by an enormous loss of mineral salts, with important digestive disturbances, the principal indication is to resort to a nutritious diet, rich in mineral salts. The phosphates should be contained in the food and then are better assimilated.

Contrary to Rehn's assertion, who states that he has obtained three cures by proper feeding, there are numerous cases in which the condition of the digestive tract was in such a state that not only could no feeding by mouth be given, but even rectal and other means of feeding were practically out of the question. The following case, due to Meslaye, is illustrative.

Female, *æt.* 14½ years. Father alcoholic, but never ill. Mother large and strong, fifty-five years old, never ill. Has had five children, the first dying in eleven days, the remaining three are well. The

patient is the fifth child. Miscarriage between the second and third pregnancy.

The patient was breast fed up to the age of twenty-two months; she walked at one year and dentition was normal. She has had measles.

When about thirteen she began to complain of pain in the lower limbs, especially after walking and principally in the knees although the joints were apparently normal. Eight or ten months after these symptoms began, rest in bed and sulphur baths were ordered. One month after this treatment there was a very marked genu valgum necessitating bilateral osteotomy. Four months later the child could walk with crutches.

Soon afterward the patient was obliged to remain in bed, the arms became heavy, certain movements were difficult to carry out, especially those involving the scapular muscles, so that she could no longer use the crutches. Four months later a kypho-scoliosis developed and in three months had made extremely rapid progress.

At the same time the ends of the fingers became deformed—drumstick shape—the legs were bent on the thighs, extension became impossible although flexion was partially possible. The patient was again admitted to hospital.

*Status.*—Child rather large for her age; very intelligent and of happy disposition.

There was no deformity of the skull or face, teeth good, roof of mouth regularly developed.

Right kypho-scoliosis forming an almost angular projection. The entire thorax is twisted and inclined to the right; sternum strongly projected forward. The ribs were vaulting at their point of insertion with the cartilages. Compensation curves bent the entire spine in different directions. The scapulæ were spread from the middle line; pelvis normal.

All the movements of the shoulder were limited and difficult. The sacro-lumbar muscular mass was very atrophied, the trunk immovable. Movements of flexion and extension were present only in the coxo-femoral joints.

The arms were thin, especially in the muscles of the forearm and hand. However, all the movements of these muscles were easy. The bones of the upper limb appeared to be normal. The phalangettes

presented a peculiar symmetrical deformity; they were in forced extension and almost dislocated. No vasomotor disturbances; movements of prehension of the hands easy.

Movements of the coxo-femoral joints easy and indolent. Complete extension of the knees impossible although the joints were intact. Flexion was easy. Extension could not be made beyond a right angle and further attempts to carry it out caused severe pain.

The ends of the bones appeared to be enlarged. The sural triiceps and adductors appeared to be atrophied on each side. The tibias were incurved inwardly. The muscles of the legs were all atrophied. The plantar arch was preserved. All the movements of the feet were possible but weak. No deformity of the toes. No disturbance of sensibility, no vasomotor phenomena. All the reflexes excepting the patellar, which was impossible to obtain, were normal. All the viscera normal. The child has never menstruated.

From the time patient entered the hospital in March, she continued to complain from time to time of pain in the thighs and legs. The pain was fixed in the bones, and was increased by pressure or movement. They lasted for several days at least and then diminished only to recur in a few days.

In raising up the child a fracture of the right thigh ensued which did not unite.

On May 15, she had a sore throat—not diphtheritic—and was transferred to another ward where, upon arrival, a fracture of the left thigh was found, symmetrical with that on the right. Five days later, when the temporary splints were removed abnormal mobility of the right tibia was discovered below disc of the tibia. The patient was put in Bonnet's wire splint and from this time on the lower limbs became rapidly deformed and an enormous œdema developed.

The lower limbs considerably shortened and all the deformities of the skeleton became more and more accentuated. The slightest movement was painful in the upper and lower limbs and jaw.

The muscles of the neck became in turn involved so that the child could not turn the head. Rapid atrophy of the muscles of the upper limbs. The humerus and forearm became incurved. Towards the end of February the general condition became serious, the pyrexia

followed by a very slight improvement. The child's stature had decreased thirty centimetres in one year.

Feeding became difficult on account of vomiting. Intelligence complete. The patient died on May 12 in marasmus after having presented abnormal mobility of the arms during the last few days of life.

Since the first publication by Bossi, of Genoa, in 1907, relating a case of osteomalacia cured by adrenalin, quite a few cases have been recorded in different countries, either successful or unsuccessful. The results of this treatment can be thus summarized: Arrest of the evolution, cicatrization and consolidation of the bone, straightening of the deformities, disappearance of the pain and an almost complete recovery of functional capacity. I shall here give an illustrative case due to León Bernard and will conclude this paper with a summary of this clinician's treatment of osteomalacia with suprarenal extract.

When Bernard first saw the patient she had been submitted without success to a number of treatments, including arsenic, Hg., continued extension, thermotherapy, electrotherapy, etc., so that it was decided to try adrenaline, a sterile 1:1000 solution of the hydrochlorate in ampoules being employed, one cubic centimetre being given subcutaneously every second day, for three consecutive months.

At the end of the three months, as the menses had greatly diminished, the exhibition of the drug was stopped during menstruation. On account of the improvement at the end of nine months' treatment an injection was only given every third day but after twelve had been given it was deemed necessary, on account of the return of the pain, to give the drug every second day and this was continued without interruption—excepting during the menses for ten days—hence making in all about eight or ten injections each month.

At the end of treatment, 183 c.c. of the adrenalin solution had been injected. The immediate effects of the injection amounted to nothing more than a slight burning sensation; fifteen minutes after the injection and lasting for about two hours there were cardiac palpitation and tremor of the limbs. The blood-pressure taken with Pachon's instrument increased one degree; no change was noted in the heart sounds.

The action of the adrenalin on the patient was really marvellous and after the thirtieth injection improvement was already appreciable; the pain was less severe, the limbs could be moved a little, the thumb and index of the left hand, the right auricular, and left arm, which were the first to be involved in the process were no longer painful, while the commencing tumefaction subsided. Then nine months after this improvement the patient commenced to raise the feet off the ground and from this time on progress was uneventful. A month later she could walk unaided and the next month she could sit and get up without assistance. At this time she had been given 82 injections.

The following month she could go down a few stairs and a month later she had recovered almost all her movements and slept well. At the same time the stature increased and the patient had regained her normal physiognomy. One hundred injections had been given at this date.

Treatment was continued for one more year although recovery seemed to be complete.

At present the face has become elongated and oval, the neck has increased in height and supports the head erect. The thorax has risen from the pelvis and the patient has now her former height. There is no pain and she gets about easily although she tires easily. The gait is a little waddling, this being due to a shortening of the left leg from fraeture.

No swelling of the bones can be detected and no callus can be found at the point of the former tumefactions; the incurvation of the fingers and left forearm still exists but there is no swelling or pain. The bones can be palpated and are hard. These deformities are, for that matter, decreasing steadily although gradually.

According to Bernard, one cubic centimetre of a 1:100 solution of adrenalin hydrochlorate should be given subcutaneously, at first every second day and according to the patient's tolerance and the reactions of the lesions to the treatment the injections can be given oftener or less frequently. At all events it is well to stop the injections every third week and in the female to suspend treatment during the menses.

The immediate effect of the treatment varies, in some cases it is

*nil.* They usually consist of some cardiac palpitation, generalized tremor and a slight and fleeting rise of the blood-pressure. But it would seem that some patients are quite intolerant and the treatment must be given up, because immediately after the injection serious accidents develop, such as pyrexia with tachycardia, small irregular pulse with a tendency to collapsus, acceleration of the respiration, cardiac distress and mydriasis.

As to remote effects none have been observed. The osteomalacia may or may not retrogress but no lesion as a result of the treatment has been so far recorded. According to Bernard the negative cases have usually been those in which the treatment was not carried out for a sufficiently long time, hence its efficaciousness should not be invoked.

Adrenalin should never be given intravenously, because experimentally it has been shown that the drug may cause atheroma of the aorta. In opposition to Josué's assertion that adrenalin should never be given more than twelve times, the cure of osteomalacia—as shown for instance in Bernard's case—should on the contrary be carried out for months or years.

Therefore, in accord with Bernard, in osteomalacia, a rapid and timid trial of the drug as recommended by some should never be countenanced, but the medicament should be exhibited with all due clinical prudence. The irregularity of the therapeutic results obtained pleads in favor of the diversity of the pathogenic factors of osteomalacia.

# SOME OBSERVATIONS ON THE SYMPTOMS AND TREATMENT OF CONGENITAL SYPHILIS IN THE SPECIAL OUT-PATIENT CLINIC\*

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DURING the past year and a half the Children's Hospital of Washington has been operating a clinic for the treatment of congenital syphilis. In this work it has fallen in line with other modern institutions in grouping for study and in administering to these unfortunate infants and children.

A few hospitals have been doing some such work for a number of years, but these clinics as yet are rather unique, and it is to be hoped that before long all institutions concerned in the treatment of children will be able to provide clinics in which luetic children may receive the thorough and intensive treatment similar to that which is given in cases of acquired lues in the adult. In this way alone can we hope to properly cope with this serious and ravaging infection.

The method of simply treating an individual case (haphazardly) while in the hospital, and occasionally giving a treatment in the Out-patient Department is ineffectual, unsatisfactory, and may at times prove harmful in that it leads the parents to feel a false sense of security in believing that the child is cured. These children require thorough and prolonged intensive treatment in order to bring about a cure, and unless such is undertaken in a properly organized clinic the results are not nearly so satisfactory. We can speak from experience, since we have tried both methods. At best it is difficult to secure coöperation on the part of patients and unless there is regularity

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\* From the Congenital Syphilis Clinic, Out-patient Department, Children's Hospital, Washington, D.C., and the Pediatric Department of Georgetown University School of Medicine.

in attendance over a long period of time, the best that can be hoped for is a relief of symptoms from time to time. But with a properly organized clinic, interest is stimulated because parents realize that something is being done for their children and they also are made to realize that they are not being discriminated against—other children are undergoing the same treatment for the same disease. The distinctly beneficial results of such treatment are seen by parents in other children, and this is decidedly helpful and stimulates a desire on their part to see the same improvement in their own children.

#### THE ROUTINE OF THE CLINIC

Our clinic is held every week and all cases of lues in the hospital and Out-patient Department are referred for treatment. All cases of suspected lues, after having been thoroughly examined in the Medical Clinic are referred, along with an opinion on the clinical manifestations, for serological tests. In this manner we secure all the evidence in each case before commencing treatment. Whenever possible a Wassermann reaction is done on the mother's blood.

A graduate nurse, under direction of one of us, prepares a solution of neo-arsphenamine and two assistants are present to hold the patient in proper position for intravenous therapy. With such a system it is easy to treat a considerable number of patients in a comparatively short time.

All children are weighed each week and careful progress notes are inserted in the records, special attention being given to

1. The behavior of apparent lesions;
2. The appearance of new symptoms;
3. The reaction from administration of the drug.

A social service worker visits the homes, records all conditions found, and notes any reactions which occur after the children leave the clinic. We have also secured volunteer workers who bring children to the hospital in their automobiles for treatment. This has proved advantageous especially in those cases which would of

necessity forego treatment because of inability to be brought by parents to the clinic—usually for domestic reasons.

In a Class-A hospital the organization and operation of such a clinic requires only the initiative of those interested, and the value of the work to this class of patients as well as to the development of therapy is inestimable.

#### THE WASSERMANN REACTION

We have had during the past year 142 cases of suspected syphilis referred to our clinic. Of this number 57 cases were treated; 53 (or 37.3 per cent.) of the 57 cases treated gave a positive Wassermann reaction with clinical manifestations of syphilis. The remaining four cases were clinical syphilis with negative Wassermann, all of which, however, showed a positive Wassermann reaction after treatment.

Out of the 53 positive cases all but 8 showed complete positive reaction; these 8 showing 1 to 3 plus. This tends to show that there is an inclination in infants and children to complete positive reaction. We emphasize this point because we feel that it is the belief of some that a Wassermann reaction in an infant or child is not as important and does not have the same relative value as a positive reaction in an adult.

Several children in spite of thorough and active treatment still show positive Wassermann but are clinically improved.

No cases are treated on the result of the Wassermann reaction alone; the serological test is considered along with evidence of clinical syphilis or syphilitic manifestations in the family. We do not usually consider a positive Wassermann reaction in the absence of other clinical findings, as positively diagnostic of syphilis or sufficient reason for subjecting the patient to treatment.

#### INVOLVEMENT OF THE NERVOUS SYSTEM

Twenty-three lumbar punctures were done on separate children. Of these, 2 were plus-minus; 1 was single plus; and 1 was completely positive. The remaining 19 were negative. The cell count varied

from 1 cell, which was the lowest, to 80 cells, which was the highest; 3 cases showed increased cell count.

Our experience from a limited number of cases suggests that involvement of the nervous system is more common than we have been able to demonstrate by abnormal findings in the cerebro-spinal fluid. We do not believe that a lumbar puncture should be performed without keeping the child in the recumbent posture for 24 hours. It has been our practice to keep all runabout cases in the hospital following a lumbar puncture, and we have had no bad effects. Infants in arms are kept in recumbent posture and taken to their homes following treatment.

We feel that it will be of interest to note the clinical symptoms in these cases and the percentages in which they occurred:

## ANALYSIS OF SYMPTOMS

	Cases.		Cases.
Snuffles .....	19	Anæmia (apparent) .....	2
General adenopathy .....	12	Mental changes .....	2
Saddle nose .....	10	Eczema .....	1
Condylomata .....	7	Perforation of soft pallet .....	1
Mucous patches .....	6	Hutchinson's teeth .....	1
Desquamation of skin .....	11	Unequal pupillary reaction .....	1
Enlarged spleen .....	5	Leukoplakia .....	1
Superficial ulcerations .....	5	Icterus neonatorum .....	1
Maculo-papular eruptions .....	6	Nystagmus .....	1
Stubborn colds .....	3	Enuresis .....	2
Keratitis .....	3	Conjunctivitis .....	1
Pseudo-paralysis .....	3	Hemorrhagic diathesis .....	1
Dactylitis .....	3	Headache .....	1
Herpes .....	3	Perforation bridge nose .....	1
Poor nutrition .....	6	Pain in legs .....	1
Enlarged head .....	4	Dyspnœa .....	1
Enlargement of epitrochlea .....	3	Sore throat .....	1
Bone changes .....	2	Hoarseness .....	1
Enlarged liver .....	4	Nervousness .....	2
Prematurity .....	2	Intertrigo .....	2
Swelling of eye lids .....	2	Arrhythmia .....	1
Arthritis .....	5	Discharging ear .....	1
Pustular skin lesions .....	2	Exophthalmos .....	1
Convulsions .....	2	Exudative lesion of scalp .....	1
Serpiginous eruptions .....	1	Rickets .....	5
Spasticity .....	2		

From the above it will be seen that skin lesions are the most common clinical findings:

#### THE INCIDENCE OF CLINICAL SYMPTOMS PER 100 CASES

	Cases.	Per cent.
Skin lesions .....	48	84.2
Snuffles .....	19	33.3
Nervousness .....	15	26.3
General adenopathy .....	12	21.
Joint and bone changes .....	11	19.2
Saddle nose .....	10	17.5
Poor nutrition .....	6	10.4
Enlarged spleen .....	5	8.7
Enlarged liver .....	4	7.
Enlarged epitrochlea .....	3	5.2
Dactylitis .....	3	5.2
Keratitis .....	3	5.2
Prematurity .....	2	3.5
Anæmia (apparent) .....	2	3.5
Perforation of soft pallet .....	1	1.7
Hutchinson's teeth .....	1	1.7
Icterus neonatorum .....	1	1.7
Perforation bridge of nose .....	1	1.7
Hoarseness .....	1	1.7
Discharging ear .....	1	1.7
Exophthalmos .....	1	1.7

In the family history of the above children 19 (or 33.3 per cent.) showed definite or very suggestive evidences of syphilis, which includes miscarriages, still-births, prematures, insanity, and extreme nervousness.

#### THE TECHNIQUE OF INTRAVENOUS TREATMENT

We have given 56 courses of intravenous treatment, consisting of neo-arsphenamine. Neo-arsphenamine is used because of the ease and rapidity with which it can be prepared, as well as injected. It is less toxic than arsphenamine. It can be administered into any available vein in concentrated solution and when given in proper dosage no proof exists that it is not as efficient as arsphenamine.

Our method of preparation is as follows: Freshly distilled water

is used exclusively, and is usually prepared the morning of the day on which it is to be used; 9-10ths of a gram of neo-arsphenamine is dissolved in 60 c.c. of this water with as little shaking as possible, and then only in a rotary manner. Only a sufficient quantity is made for immediate use and it is rarely necessary for the solution to be prepared for a period longer than 45 minutes. The water is always cool (room temperature) and kept in a cool place away from the room in which the administration is carried on. Emphasis is laid on this method of preparation because, according to the experiments conducted in the Public Health laboratory, toxicity increases in direct proportion to the amount of shaking and the length of standing of the solution.

The child is weighed each week upon admission to the clinic and its weight is figured in kilograms. It has been our custom to administer 15 mgs. of neo-arsphenamine, which is contained in 1 c.c. of the solution, for each kilogram of body weight. The age is not taken into consideration; gradation of doses being calculated entirely by the weight of the patient.

Weekly injections of a 1 per cent. solution of bichloride of mercury are given. The dose of this solution is half a minim per kilogram of body weight, each half minim representing approximately 1-180th of a grain of mercuric chloride. These injections are made into the muscles of the buttocks. By the use of intramuscular injection of the drug we are certain that the child receives the proper dosage. We have also used inunction and mercury orally. We are not favorably impressed by the oral administration in children. One child took 50 half-grain tablets of mercury with chalk at one dose and showed no ill effects. We have had some induration following these injections of mercury, but have never seen any signs of infection or sloughing.

Any available vein is used for the administration of neo-arsphenamine, the one of choice being at the bend of the elbow; secondly, a vein in the scalp, which we usually find to be quite prominent and readily entered; thirdly, the external jugular vein, which can be entered in practically every case without any particular difficulty

and is nearly always available when the others for some reason are blocked or too small to be used. In no case has it been necessary to cut down on a vein, and it has never been necessary to forego treatment for the lack of a vein.

When using the vein in the arm it has not been our custom to use a tourniquet, *per se*. After wrapping the child in a sheet to prevent wriggling, the nurse or attendant grasps the arm tightly well above the elbow and in this manner the vein is sufficiently blocked to become filled with blood. As soon as the vein is punctured the attendant simply releases the pressure and the injection is commenced. This relieves the necessity of waiting until the tourniquet is released and the annoyance of having the needle displaced by wriggling of the child, which often follows in attempting to remove the tourniquet.

Our methods in using the scalp and jugular veins are best realized by an examination of the accompanying photographs.

We mention the longitudinal sinus as a method for giving intravenous neo-arsphenamine only to condemn the procedure. We have never found it necessary to use this channel in our work, nor do we believe it justifiable. One case in which this procedure was followed was autopsied by one of us (M.S.) and it showed that this sinus had been transfixated by the needle, with blood in the brain tissue (this case was not treated by either of us).

Injections can be made rather rapidly under close observation for any untoward symptoms. We have never seen any children showing such symptoms during injection or immediately following and we have not been forced to stop the administration of the drug for this reason.

It has been of interest to note a number of cases where small amounts of the solution escaped into the tissues and in which no reaction occurred. If it is noticed that the solution is not flowing freely or any swelling of the tissues is noticeable, injection is stopped in that particular vein and a new vein is used.

The intravenous injections of neo-arsphenamine are given in 6 weekly doses followed by a two months' course of mercury. Mercury injections are also given during the course of neo-arsphenamine.

FIG. 1.



Injection through the median vein or in its branches at the bend of the elbow. Children's Hospital, Washington, D. C.

FIG. 2.



Injection through a vein in the scalp in the older child. Children's Hospital, Washington, D. C.

FIG. 3.



Injecting solution into external jugular vein. Children's Hospital, Washington, D. C.



## THE UNTOWARD RESULTS IN 350 INJECTIONS

In 350 injections of neo-arsphenamine we have seen:

- 2 cases in which we believe irritation of the kidney occurred;
- 1 case became stuporous after lumbar puncture and intravenous treatment, but soon reacted (severe case);
- 2 cases of pain about the heart; one was accompanied by tachycardia and the other became dyspnoëic (these reactions occurred several days after the drug was given);
- 1 case with palpable thyroid, with fine tremor of the fingers.

We presume that these effects were due to the use of the drug, but we could not be certain. Several children vomited immediately following the injection, and some mothers reported that their children vomited the day following the injection. We have not been able to follow the reactions as closely as we desired. These effects were not lasting and treatment in all cases was resumed.

We regret that our cases were not regular in attendance and that many of them stopped coming to the clinic before completion of treatment. In spite of this, however, we have seen only 2 cases in which there has been no definite improvement.

We are unable to comment further upon the real value of the treatment outlined above except to state that if carried out regularly, it is distinctly beneficial. Besides the specific therapy it is essential to carefully supervise the feeding and general management of these patients. No line of treatment can be successful if this is overlooked. The methods we have detailed have been used successfully by Jean, of Washington University, for a period of eight years.<sup>1</sup>

In closing we wish to emphasize:

1. The frequency of the complete positive Wassermann reaction occurring in children.
2. The frequency of a normal spinal fluid in our cases—19 out of 23.

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<sup>1</sup> JEAN P. C., *American Journal, Diseases of Children*, August, 1920, "Review of Literature on Syphilis in Infancy and Childhood," p. 132.

3. The absence of severe reactions following injection of neo-arsphenamine.

4. That intravenous therapy in infants and children requires only such practice and skill as can be readily acquired.

5. That lesions most rapidly affected in children and infants are those of the skin and mucous membrane.

6. That we condemn the use of the longitudinal sinus as an entry for administration.

7. That the good results in the children who have attended the clinic regularly lead us to believe that this method is very efficient in the treatment of congenital syphilis.

8. That we believe consistent and persistent treatment is the keynote of success in the treatment.

9. That the general management must be carefully supervised and the state of nutrition of these children improved in order to secure the best results.

**THE INFANTILE THORAX OF THE RACHITIC  
CHILD AS A PREDISPOSING FACTOR  
IN BRONCHIAL INFECTIONS \***

**CLINIC OF DR. JOHN FOOTE**

Children's Hospital, Washington, D.C.

You have been told that rachitis, or rickets, is an important predisposing condition in the bronchitis of young children. You may remember that the patient presented to you last week, a rachitic negro child, had been admitted to the hospital because of cough and fever from which he recovered very promptly.

The patient before you to-day, a negro male child, aged two, is acutely ill with a respiratory infection, characterized by cough, rapid respiration and a temperature fluctuating between 100° and 103.6° F. You will observe that this child presents the classical picture of rickets—the square head, round thorax, out-flaring ribs, and the depression on either side of sternal articulation called Harrison's groove. Note also the marked tibial curvature, especially in the right leg. This child has never walked successfully. Although he is two years of age his weight is only 15½ pounds, while the normal weight of a normal male child of this age should be at least 25 pounds. The history shows that he was breast fed for only a short period, as his mother was obliged to go out to work. He was raised in a tenement district, unquestionably with few hygienic advantages. The history relates that he has always been "poorly," had frequent attacks of diarrhœa last summer and was subject to colds.

The present illness began a few days before admission to the hospital. His temperature on admission was 104.4 by rectum and he was much distended. Relief of abdominal distension produced a prompt drop in the temperature. His cough is persistent, but he takes a weak whole milk formula with avidity and does not vomit. His respirations are 40 to the minute; his pulse, 130. There is some retraction of the soft parts under the costal arch when he breathes, and the alæ nasi are active. His urine is normal and his leucocyte

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\* Given before the Third Year Class of Georgetown University Medical School, March 30, 1922.

count is 14,600, with a differential count that shows a slight relative neutrophilic increase.

Examination of the chest reveals a hyperresonant percussion note anteriorly below the sixth rib due to the distension of the stomach and intestines. The costal cage flares out so that the angle formed by the lower ribs with the sternum is even less acute than in the newborn. The percussion note is also tympanitic behind on both sides of the spine from the sixth rib downward, and it will be noted that the ribs diverge from the spine at almost right angles. No deviation from normal will be found in the percussion notes elicited elsewhere in the thorax.

The stethoscope reveals some soft bubbling râles heard on inspiration over the entire upper chest, but especially marked in the second and third interspaces anteriorly near the sternum. Posteriorly, below the angle of the right scapula are heard a few crepitant râles, and the expiration seems very slightly prolonged. The heart sounds are rapid and arrhythmic, and the pulmonary second sound is accentuated. Breathing is entirely of the abdominal type. The child cries loudly after coughing.

#### THE NEO-NATAL TYPE OF THORAX

We are dealing here with a very common condition—bronchitis in a rachitic child, or in a child, who because of rickets, has the infantile or neo-natal type of thorax. Practically all of the children with rachitis who come to this hospital, are entered because of an acute respiratory infection—bronchitis or broncho-pneumonia. Many hypotheses have been offered in an attempt to account for the well-known susceptibility of these children to bronchial infection, but only one of these has an obvious physical basis. No one can escape the fact that these rachitic children who are susceptible to bronchitis and pneumonia do not possess a thorax conforming to the type of normal children of a similar age, but rather one that resembles the thorax of the newborn child. It might be even said that they exaggerate the characteristics of the thorax of the newborn.

#### SOME FEATURES OF THE NEO-NATAL THORAX

It will be remembered that the thorax of the newborn infant is round in its outline when measured at the nipple, and that as child-

FIG. 1.



The thorax of a 3-months-old child, showing a slight tendency toward normal angulation of ribs at their junction with the spinæ. Children's Hospital, Washington, D. C.

FIG. 2.



The thorax of an 8-months-old child suffering from rickets of moderate degree. Note that the ribs come off from the spine at a right angle as in the embryo.

hood is reached it gradually becomes more elliptical. Thus, while the antero-posterior diameter bears the relation to the transverse of 1 to 1.5 at birth, it is found in the adult to be 1 to 3. The newborn child had a "chesty" appearance as a result, which disappears before he is three years old, when the thorax has become twice as wide as it is deep. The ribs, too, come off from the spinal column at a more acute angle as time goes on, the level of the anterior surface of the chest as measured by the relation of the angle of Ludovici to the vertebræ is depressed, and the lower ribs slant off from their articulation with the sternum, until the triangle which they form in the epigastric region causes a disappearance of their tendency to flare out at the sides.

The structure of the bones in the thorax at birth, too, is different from that of the child of three or four. The ribs are soft and the sternum is made up of separate unossified pieces of cartilage. The muscles are weak and are not able to contract strongly, since their attachments are flexible and not rigid. The infant's cough, therefore, is feeble and he is not easily able to expel mucus.

In addition, since the development of negative pressure in the pleural cavity of the infant depends on the growth of the thorax and its transition from a largely cartilaginous to a bony structure, it must be seen that any portion of the lungs under these conditions must easily collapse either partly or wholly if for any reason a bronchus should become plugged up through inflammatory exudate or from any other cause.

We do not need to look at this rachitic two-year-old child very carefully to see that his thorax has all the characteristics of the chest of the newborn child. It is round, not elliptical; it is short from the axilla to the lower ribs; the lower ribs flare outwardly; the abdomen is prominent.

Since the changes which take place in the shape of the thorax from infancy to adult life are due to the pull of the muscles against fixed points furnished by the rapidly calcifying bones, and since the bones in the rachitic child are soft and the muscles are flabby, it is easy to see why the thorax of the child suffering from rachitis does not develop into the type of thorax possessed by a normal child of similar age.

## LOWERED BRONCHIAL RESISTANCE

It is easy also to see in this condition at least one plausible explanation for the lowered bronchial resistance of rachitic children. The ability to expel irritating or offending material which is a normal attribute of the normal child with hard ribs and firm muscles, is not possessed by the rachitic child. The lack of sufficient negative pressure in the pleura, with its tendency toward incomplete alveolar distension is another reason, easily suggesting itself, as a cause of the decreased resistance of the bronchi of these rachitic children toward infection.

Another, and apparently a logical reason why the bronchi of these children do not react normally against irritation and infection, is in the discovery of the existence among some rachitic children of a tendency toward the condition known as broncho-tetany. This was, I think, first described by Lederer about eight years ago. He found a tonic spasm of the muscles of the bronchi in six children among fifty-eight who had spasmophilia. All of his patients, however, were under six months of age. As many rachitic children present symptoms of spasmophilia, there is excellent reason to assume that the bronchial musculature in this disease is also frequently potentially spasmophilic, and liable to retain secretion in the terminal bronchi to a pathological degree.

## THE BRONCHO-PNEUMONIC TENDENCY

The extension of a so-called capillary bronchitis to a broncho-pneumonia is of such frequent occurrence that it is safe to assume some involvement of the alveoli in any long continued bronchitis accompanied by fairly high fever. The work of Miller, showing how the alveoli bud off into sacculations even from the sides of the terminal bronchiole and atrium, shows that no sharp line of demarcation can be drawn between infection in the smaller bronchioles and broncho-pneumonia. The same factors which produce bronchitis in a child may well produce broncho-pneumonia; both have the same etiology.

## TREATMENT AND MANAGEMENT

The rachitic child suffering from bronchitis should have even more careful hygienic care than the average child with the same

disease. In choosing a room for the patient, let it be required that it have plenty of sunlight. The temperature of the room should be about 70°F., and it must vary as little as possible. Care must be taken with the rachitic child to reduce the tendency toward abdominal tympanites by giving a daily enema of a small quantity of soapsuds, which should be drained away by a rectal tube while at the same time employing gentle massage of the abdomen.

One danger that must be avoided is the giving of opiates to diminish coughing. Coughing should be permitted to a degree not usually advised in other forms of bronchitis, since the drainage of the bronchi in this type of chest is usually inadequate. The use of a thoracic-abdominal binder may help to make the expulsive effort more effectual.

The food should be ample, of a liquid or semi-solid nature, given at intervals of three hours. The employment of cod-liver oil or the giving of quantities of cream at this time is not advisable, as the digestion and absorption are both impaired. But when the bronchitis has subsided and normal diet can be resumed, the administration of cod-liver oil should be begun, giving about one drachm daily to each ten pounds of weight, dividing this into three doses and giving it at regular feeding times. The maximum total amount in twenty-four hours should be one-half an ounce. Sunshine and cod-liver oil will cure rachitis provided the patient takes in addition a well-balanced diet. The sunshine should not be indoor sunshine, but the direct rays or reflected rays of the sun, which have not been passed through glass. The reason why negroes, Italians and other races with pigmented skins living in Northern countries, are subject to rickets, may lie in the fact that they possess in their skins a natural barrier against the chemical rays of the sun, and living under conditions in this country where they have little exposure to direct sunlight, their body tissues suffer from this helio-insulation.

Certain it is that those who are able to live much out of doors, whether children or adults, have little to fear from respiratory infections. Bronchitis and rickets both thrive and fatten on those who are obliged to live indoors overmuch.

## ASCITES IN INFANTS

WITH REPORT OF A CASE

BY C. A. SCHERER, M.D., F.A.C.P.

Duluth, Minnesota.

THE literature on ascites in infants has not been greatly augmented since the very complete review by Cowie in 1910. This review includes nine cases which Cowie divides into two groups according to age. Group 1 includes six cases in nursing infants and Group 2, three cases in later infancy. In the latter group several cases were true chylous ascites due to obstruction of the thoracic duct. Notable among these is the case reported by Morton in 1691. This case, in a child of two years, was due to the compression of the chyle duct by enlarged (tuberculous?) bronchial nodes with resultant leakage from the receptaculum and the lacteal vessels! In the first group the records show that four of the cases made complete recovery while in two the outcome is not noted. The cases of Wilhelms (1874) and Winniwater (1877) both had demonstrable abdominal tumors and true chyle. The four cases that recovered had no demonstrable lesions and the fluid was not definitely proved to be chyle.

Complete search of the available literature shows five further cases, of which four fall into Cowie's second group and only one in the first. Of the former, the case reported by Huber and Silver is of interest because of the excellent result of the operative procedure. Laparotomy revealed very greatly enlarged lacteal vessels of the intestine and mesentery and enlarged mesenteric lymph-nodes. The transudate was completely removed by internal drainage with imbedded strands of coarse silk.

Gandin reports a case of large mesenteric cyst which contained a chyle-like fluid. Numerous cases of cysts of the mesentery and even of the omentum have been reported.

Cattanea reports two cases of ascites in girls of four and four and

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\* Read before The Northwestern Pediatric Society.

a half years. One of these was tuberculous at the onset and the other developed tuberculosis after the ascites recurred. This latter is the only case in which there is any mention of recurrence after ascites had disappeared a year or more. In this child the fluid in the abdomen reappeared after an attack of measles and a short time later the child developed an open tuberculosis and died.

The case to be included under the first group is reported by Smidt van Gelden and, as it closely resembles the case to be reported, is abstracted here.

The fifth child, of healthy parents, born in Java, had a swollen abdomen at birth. At 2 months, puncture became necessary because of pressure symptoms, 1 litre of chylous fluid was removed. Puncture was repeated six weeks later and at intervals thereafter.

*History.*—Pregnancy and labor without incident; four other children healthy; no history of syphilis or tuberculosis, of miscarriage or premature delivery. The girl was ten months old when her parents returned to Holland, weighed 3600 grams, appeared very atrophic with swollen abdomen. No pain on palpation. Free fluid could be made out in the abdomen. Liver and spleen not enlarged. Heart and lungs normal. No enlarged glands. No œdema. Mucous membranes normal. No sugar or albumin in urine. Child was very susceptible to temperature changes. Mentally in good state. Breast fed, could drink very little without regurgitating; total daily fluid intake 600 grams. Normal stools. General condition had grown worse since birth. Three weeks after last puncture slight dyspepsia, after a few days all symptoms disappeared and the condition suddenly improved, she could drink more, the abdomen grew smaller, the face, arms and legs filled out. At eleven months the child had acute dyspepsia from which it rapidly recovered, at fifteen months the child was perfectly well.

The chylous fluid was examined three times, April, May and July, 1919, with these findings—

Fat .....	17.73%	2.81	21.7
Albumin .....	2.81	4.45	3.9
Sugar .....	0	0.5	
Ash .....	0.79	0.65	

The author's case falls in the first group. The child was four months old when the fluid first was noticed. It was removed by

repeated tapping. The fluid was at first clear and straw-colored and later milky and chyle-like. There was no demonstrable causative disease condition. There was no general œdema, and the child recovered completely.

Alice H., born October 29, 1920, first child, first pregnancy. B.W. 2810. Father, age 36; mother, age 34; both well and strong.

Child was full term; well-developed, active infant; examined in our routine examination of the newborn.

After a rather stormy nursing period of three weeks the child was taken from breast temporarily because of severe breast abscesses after which lactation could not be reëstablished. The infant did not do well on a whole milk mixture and following a severe disturbance of digestion was put on protein milk, on which it regained its loss and after six weeks was again fed whole milk, oatmeal water, dextri-maltose mixture. Weight at two months 3665 g.

At three months (January 10, 1921) weight 3905 (+385), the infant was brought in for routine examination and it was noted at this time that the abdomen was slightly larger than normal. No suspicion of fluid. There was some difficulty with breathing. The stools were frequent and showed some undigested food.

January 31.—Child reported as being restless and spitting up some food. Abdomen distinctly distended, but no special examination for the demonstration of fluid in the abdomen was made. Stools were distinctly fat-soap, green and highly acid. Because the child had done very well on protein milk, which the mother was able to prepare in the home, this food was given with about 8 per cent. dextri-maltose. On February 5th, the child was brought in because of intense swelling of the abdomen and difficulty in breathing.

Examination showed the child to be in practically the same condition as when previously seen, moderate turgor, happy and bright. The abdomen, however, was greatly distended, measuring 54 c.m.; chest 41 c.m. There was also distinct dyspnœa with slight cyanosis. There was no sign of enlargement of the superficial veins of the abdomen. Free fluid was easily demonstrated. A urine specimen at this time showed a few leucocytes, a very faint trace of albumin and an occasional kidney epithelium cell. Temperature normal. White count 7,640; hemoglobin, 75 per cent.; weight, 4720 (+815).

No swelling of extremities now or at any time during the illness. Stools: Typical protein milk. Wassermann on child and both parents taken and later reported negative. An attempt to procure free catharsis resulted in six loose bowel movements in twelve hours with no noticeable effect upon the free fluid in the abdomen.

Two days later the distension became so great as to cause severe interference with the respiration and much cyanosis, with extreme restlessness and crying, and the abdomen was tapped in the right lower quadrant just outside of the rectus. A quart (1175 c.c.) of clear straw-colored fluid was removed, and the child dropped off into a quiet sleep, the first in three days. The fluid showed a few leucocytes, a moderate amount of albumin and a very faint trace of sugar. No fat at this time. Specific gravity, 1.008. No tubercle bacilli at any time. Some fluid injected into guinea-pig was sterile. Von Pirquet and Mantoux both negative.

The withdrawal of fluid had made the child so comfortable that the parents insisted on further puncture when ever the respiration became at all embarrassed, even though the dangers of infection were explained to them. As a result 350 c.c. were removed on the ninth, and 800 on the tenth. After the abdomen had been emptied as completely as possible by the removal of 760 c.c. on the eleventh, careful palpation of the abdomen was made which was entirely negative. Spleen not palpable and liver 2.5 cm. below margin of ribs in nipple line. X-ray pictures of both chest and abdomen taken at this time were negative as were also several later pictures. X-ray treatments to the reduced abdomen were given: 2 M. A., 8 inch spark gap, three to five minutes. These were repeated alternating to back and abdomen and to sides every week for seven treatments with no noticeable effect. The abdomen was also exposed to sunlight until a deep tan was obtained.

Fluid was drawn off thirty-one times, varying in amount from 350 to 1250 c.c., or a total of about twenty-four quarts from February 28th to April 28th, inclusive.

About the first of March the fluid became gradually more milky. Shaken with ether the fluid became clear and the evaporated ether left fat on the filter paper. Increasingly cloudy, on March 10th,

the fluid contained 1.45 per cent. fat by the Babcock method, and always a moderate amount of sugar. The attempt was made to prove the presence of true chylous ascites by feeding finely powdered charcoal and later carmine. These substances were not recovered from the ascitic fluid. Phenolsulphonephthalein test was unsuccessful because a large part of the urine was lost. The remainder showed 40 per cent. function. None was recovered from the ascitic fluid removed twenty-four hours after the injection. Specific gravity of the fluid at this time 1.024.

Early in April the fluid became clear and straw-colored. Fat, 0.4 per cent.; faint trace of sugar; albumin marked, and a few leucocytes. Specific gravity, 1.008.

The last puncture was made April 28th, a small sized trocar being used in the linea alba just below the umbilicus. This puncture leaked for about three days after which the abdomen was constantly soft without demonstrable fluid up to the present time.

After the first month the urine was negative, the leucocyte count within normal limits and the patient happy, active, and thriving, except when the distension caused distress. Three weeks after the last puncture with no demonstrable fluid in the abdomen the child weighed 6225 gms., a gain of 2320 in three months. The child was on equal parts whole milk and oatmeal water with cereal and fat free broth, approximately 640 calories, during the last two months.

The urine had a high chlorine content throughout the illness and just before the last tapping all salt was left out of the food in preparation for determining the excretion and retention, together with the amount of sodium chloride, in the ascitic fluid. After the abdomen had been normal for three weeks the salt was again put back into the diet without any bad effects.

The child last came under observation in January, 1922, when about fifteen months of age. She weighed 11.320 grams (25 pounds), was well nourished, active and good natured. Mother reports that appetite is excellent, sleeps all night and walks all around the house. The abdomen is slightly larger than normal but not pendulous. General physical examination shows a very good specimen throughout.

There is much confusion in the classification of the various forms of ascites, which probably arises from the attempt to base such a

classification upon the nature of the fluid alone. Recent authors reporting cases have devoted much time to the study of the fluid with the purpose of determining its origin and nature. Quincke's classification into chylous, chyloform and pseudo-chylous is contested by several authors. Some consider all abdominal transudates true chylous ascites with greater or lesser amounts of fat. Huber and Silver suggest the following points to assist in clearing up the situation:

1. True chylous ascites is due to the presence of chyle.
2. Chyloform or fatty ascites owes its milk-like appearance to emulsified fat from fatty degeneration of the cellular elements.
3. In the non-fatty form the opalescence is not due to fat in emulsion, but to some opalescent substance the nature of which is not known.

The second premise seems to be borne out by the case reported here in which the fluid, at first clear and straw-colored, became opalescent and contained 1.45 per cent. fat when the secretion was at its height. Later, as the amount secreted became less, the fluid was again clear.

Chylous and chyloform effusions were studied as to their composition and from a pathological standpoint by Wallis and Scholberg in 1910; they believe there are no characteristic pathological anatomical changes. Kelly (1907) would restrict the terms to cases presenting injury or ulceration of the chylous system. Witlin (1909) believes chylous ascites or chylothorax can be explained only by trauma. Undoubted rupture in some part of the chylous system has been reported only occasionally. Mechanical obstruction, in the course of the duct from receptaculum to left subclavian vein or in mesentery tributaries from new growths, cicatrices, tumors, etc., has been frequently observed. The chylous ascitic fluid has a specific gravity, 1.007 to 1.037; average 1.0158; chyloform, 1.009 to 1.026; average 1.01625. The latter is usually associated with new growths or chronic peritonitis. In non-fatty the specific gravity is 1.005 to 1.030, average 1.0132, its milky appearance is not lost when shaken with ether, benzol, etc. Even though fat is present the amount is too small to give rise to milky appearance. Microchemical reactions for fat are negative. Precipitation of albumin leaves a clear solution.

Gandin made a critical study of the subject and was unable to establish differential characteristics of the fluid in the various forms. His conclusions are briefly: Even small percentages of the fat (0.01 to 0.1 per cent.) of chyme or homogeneous milk will impart a milky cloudiness to a transparent fluid. The milky appearance of a serous effusion is due to emulsified fat. The presence of emulsified fat without admixture of chyle is not probable. As chyle represents the only source of finely emulsified fat in the body, milky and opalescent effusions must be regarded as chylous. The terms "chyliform" and "pseudo-chylous" are superfluous as they are not applicable to distinct pathogenetic entities. Effusions containing drops of fat (fatty or adipose) do not present typical milky appearance.

It would seem from this great diversity of opinion that the origin of the fluid can hardly be determined from its chemical examination. Nor is this determination of great value in the diagnosis. The pathological lesion is usually found only at autopsy or at operation as in Huber and Silver's case. In the series of cases reported there was no opportunity of demonstrating the pathology. In our case there was evidently no direct passage from the chyle vessels into the abdominal cavity. The question of the relation of the chlorides as a cause of the retention deserves more study.

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# **Surgery**

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## **RÖNTGEN RAY LOCALIZATION OF A GLIOMATOUS CYST OF THE BRAIN BY THE INJECTION OF AIR**

**BY CHARLES H. FRAZIER  
and  
FRANCIS C. GRANT**

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DURING the last few years the interest in the injection of air into the cranial cavity as a means of diagnosing the presence of a brain tumor has greatly increased. The work of Dandy along these lines has shown that air may be injected into the cerebral ventricles with comparative ease, though not without risk. Even before this it was known that intracranial air appeared on the X-ray plate as an area of decreased density. The plates taken following a fracture of the skull with passage of air into the cranial cavity (traumatic intracranial aerocele) proved that the ventricles and other areas could be outlined with great distinctness.

In the case we wish to report, advantage was taken of this fact to define exactly the limits of a large cyst encountered while operating on a case of suspected brain tumor. After an osteoplastic flap had been turned back and the dura opened, palpation of the cerebral cortex revealed an area yielding the sensation of fluid under tension. A cannula was passed down into this region and 25 c.c. of a yellowish fluid obtained. The presence of fluid of this description in this position is almost pathognomonic of the presence of a gliomatous cyst. There was no way of knowing how extensive this cyst was or the precise area involved without opening the cortex. While the patient's condition was in no way alarming, nevertheless the cutting down through the cortex upon a glioma of unknown size in the face of a considerable increase of intracranial pressure as shown by the tenseness of the dura, especially since it is a well-known clinical fact that in the presence of a glioma the cerebrum is very prone to undergo œdema, seemed a too formidable procedure. About 25 c.c. of fluid was therefore removed from the cyst by aspiration and an

equal amount of air injected. The wound was then closed without difficulty. The patient was sent to the X-ray department for a skiagram. The outline of the cyst and the tumor mass lying in its wall as seen in the X-ray plate is shown in the accompanying diagrams. With the tumor thus carefully localized it was a relatively simple matter to perform a secondary operation a week later, open the cortex, expose the cyst, and remove the gliomatous mass lying in its wall. The patient made an uneventful recovery. The clinical notes on the case are as follows:

S. E., white male, aged 32. Admitted to the University Hospital on the service of Dr. C. H. Frazier on 3-6-22.

*Chief Complaint.*—Headache and convulsions.

*History of Present Illness.*—Perfectly well until 9 years ago at which time he noted a sensation of numbness starting in his left knee and running down his left leg to the foot. During these attacks his left leg felt weak. These attacks came on at irregular intervals and the numbness gradually spread to the whole left side of his body—leg, trunk, arm, and at times face and head. This sensory disturbance was strictly limited to the left side of the body. Left arm and leg have gradually become weaker, particularly during the past year or two, and some spasticity and a difficulty in performing finer movements such as tying shoelace or necktie have been noted. At present he walks with a limp, swings his left leg by moving his pelvic girdle, and has difficulty in lifting his toes from the ground. The left arm is held stiff close to the body, slightly flexed and pronated, but may be easily moved. There have been 8 or 9 attacks in which the numbness and sense of weakness spread rapidly up the left side followed by unconsciousness and generalized convulsions lasting 10 to 15 minutes, during which he has bitten his tongue. These attacks began with the numb sensation in the leg, but there were no localized muscular twitchings noted during the onset. Attacks followed by severe headaches. Headache has been a constant symptom for the past 6 weeks, frontal in distribution, paroxysmal and very severe. He has also noted failing vision. No disturbance of the sense of smell or taste.

No objective symptoms which are abnormal.

No symptoms which are referable to the cardiac, respiratory, G-I or G-U tracts. No dyspnoea, cough, oedema or palpitation.

*Previous Medical History.*—Had measles as a child. Pneumonia in 1918. Denies venereal disease. Has had a running ear on the left side all his life.

*Family History.*—One of a large, healthy family. No cancer, insanity, epilepsy or tuberculosis. Wife and one child living and well. Wife had two miscarriages.

Milk wagon driver by occupation. Habits fair, personal hygiene poor.

*Physical Examination.*—The patient is a small, rather nervous looking man of about 30. He appears frail and has not a healthy color. The eyes show rather suggestive exophthalmus. The left arm is carried in partial flexion and

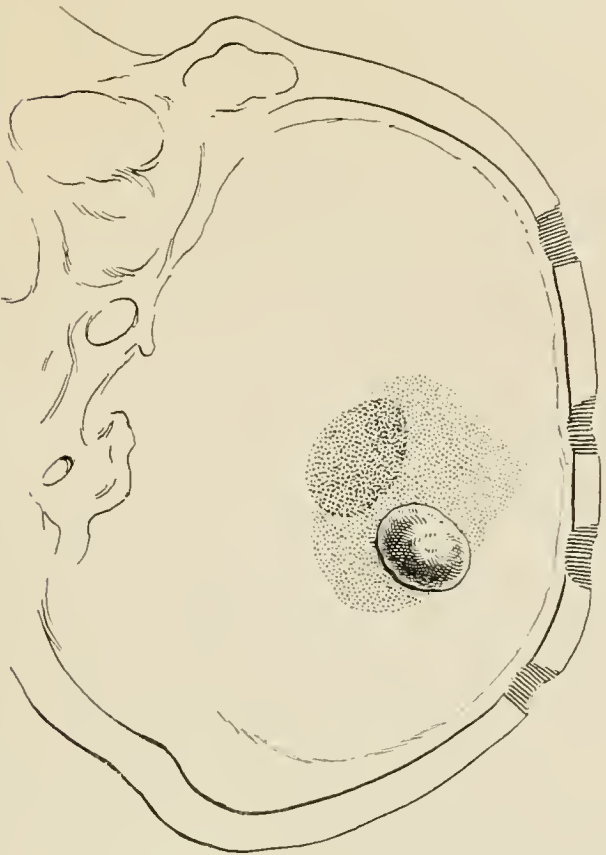


FIG. 1.

Tracing from X-ray of head following injection of air into cyst. Stippled area represents area of decreased density due to air in cyst. Filling defect caused by tumor is drawn in somewhat diagrammatically to show its exact position.

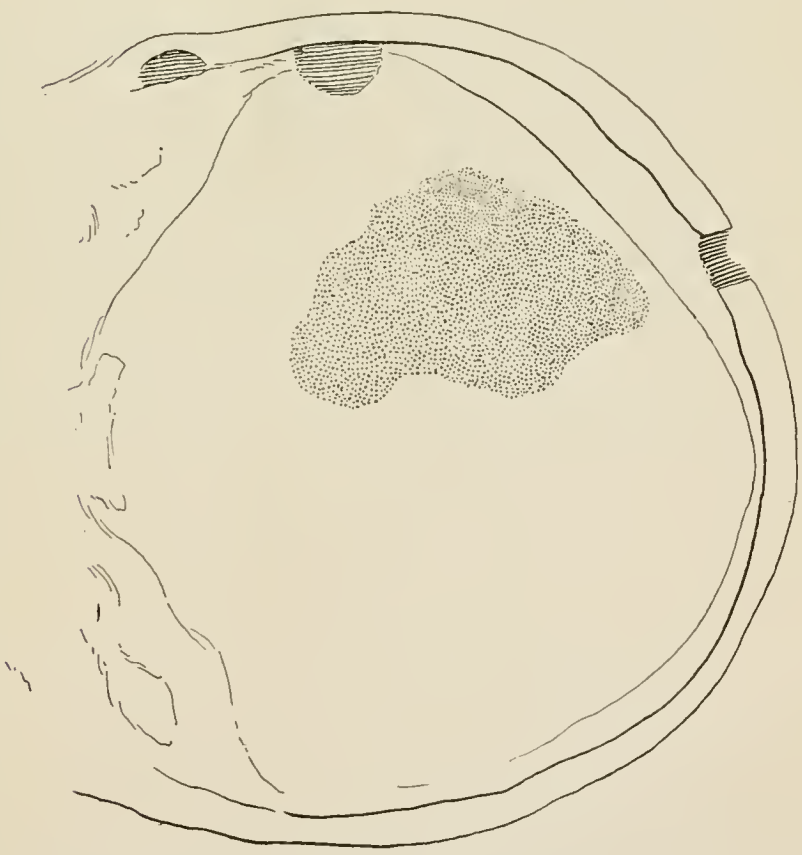
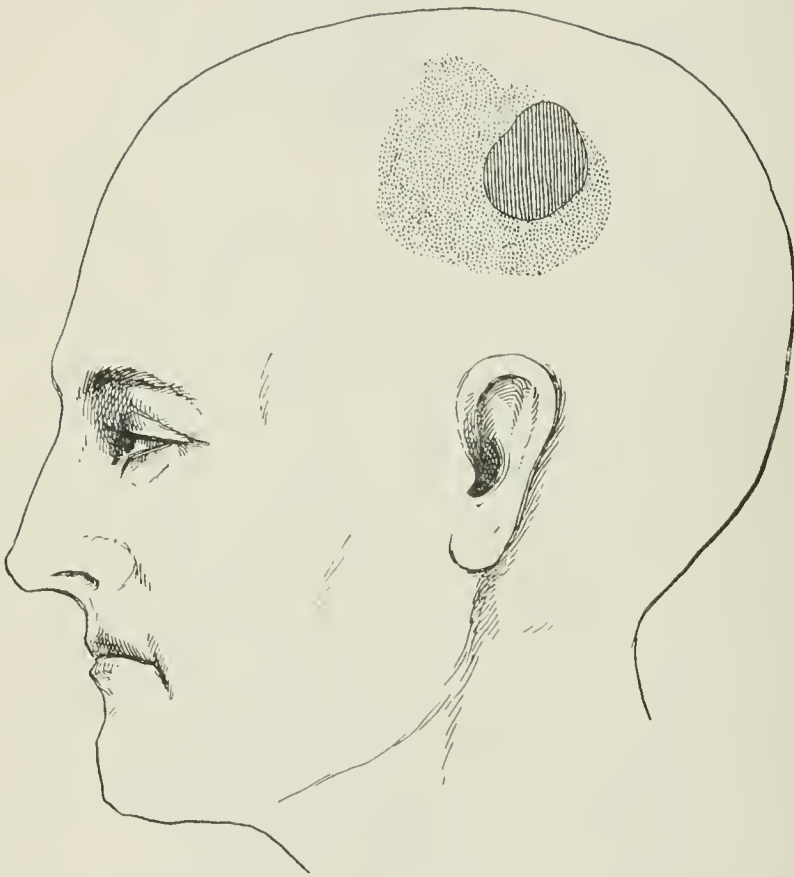


FIG. 2.

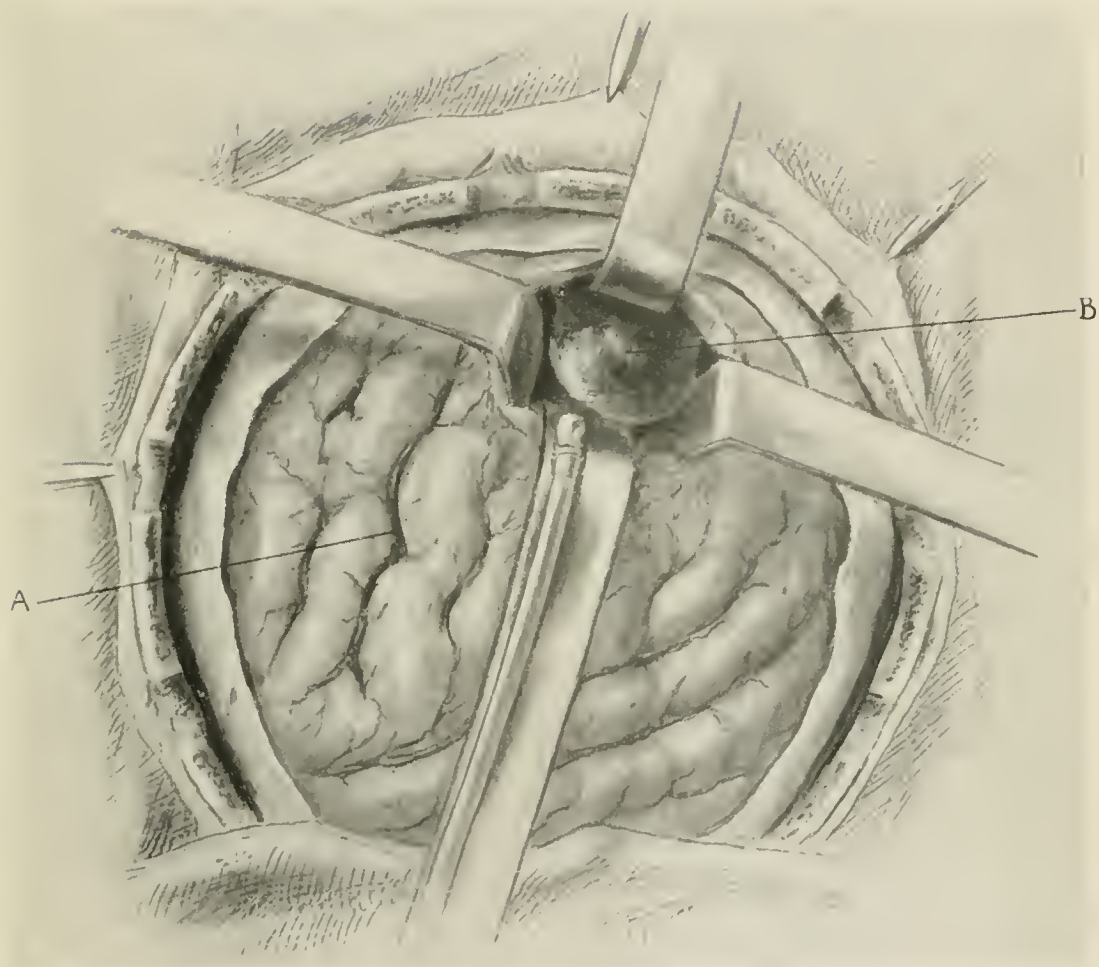
Anterio-posterior X-ray plate of head. Cyst filled with air represented by stippled area. Tumor mass did not show in this X-ray plate.

FIG. 3.



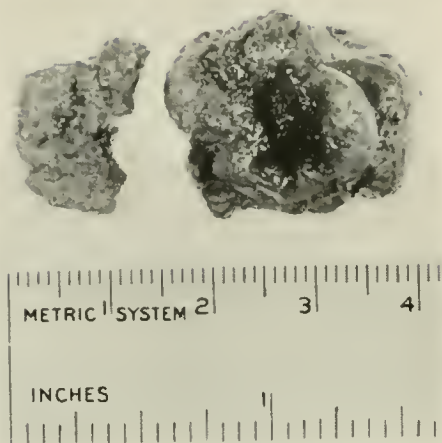
Shows exact position of cyst and tumor mass in relation to external topography of head.

FIG. 4.



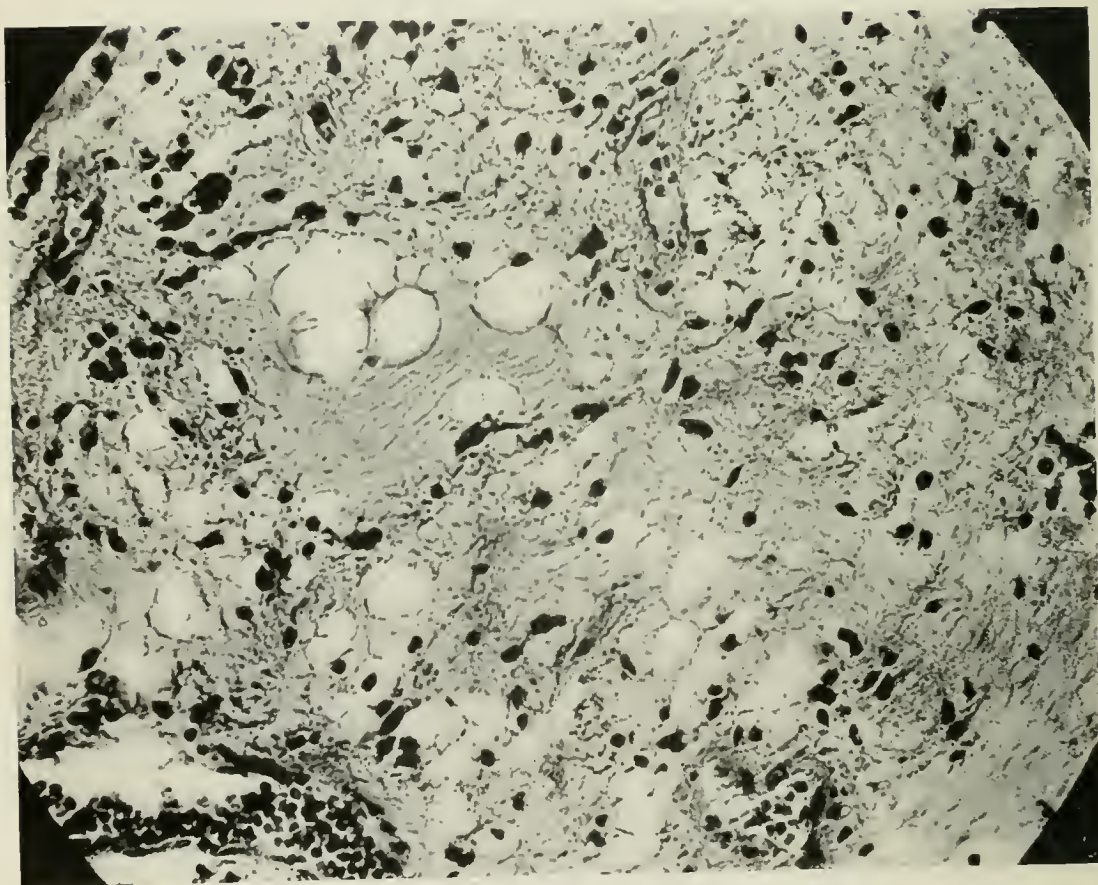
Tumor as seen at operation following transcortical incision into cyst. *A*, fissure of Rolando;  
*B*, tumor mass presenting.

FIG. 5.



Shows size and shape of tumor mass.

FIG. 6.



Photomicrograph showing gliomatous nature of tumor.

the fingers are stiff. He walks and drags the toe and left foot. The gait is extremely spastic on the left side.

The head is small and appears grossly normal. No cranial defects. Hair thick. Scalp negative. No areas of tenderness.

The nose appears grossly normal. Sense of smell is normal.

The eyes showed marked widening of the palpebral fissure and protrusion of the eyeball. The extraocular movements are full and normal. There is a suggestion of weakness of the right external rectus. The pupils are round and equal. They respond promptly to light and in accommodation. The eye-grounds show papillœdema O. D., plus 3; O. S., plus  $3\frac{1}{2}$ .

There is no gross defect in the visual field when tested with the finger.

There is no disturbance of the muscles of mastication. No sensory disturbance of the face.

There is no weakness in any of the branches of the facial nerve. The muscles respond on each side equally well.

Hearing is decreased on the left side. There is a slight discharge from this ear. No tinnitus or vertigo.

There is no difficulty in swallowing. The voice is husky and harsh. Pulse is slow and regular, 70. Respirations normal.

The tongue is very large and red with white patches over its surface. There is a fine tremor about the edges and many teeth marks along its border. It protrudes in the midline and there is no difficulty in motion.

The neck shows a marked fullness over the thyroid region. The gland is palpable and enlarged on both sides. Below the jaw and on the right side is a small mass that lies in the superficial tissues and feels like a cyst or soft gland. It is about the size of a walnut.

The chest is negative to inspection and percussion. No abnormalities noted. No disturbance of sensation. No râles or adventitious sounds.

The heart is slow, regular, normal in size. Slight roughening of the first sound. Blood-pressure, 120-70.

Abdomen negative. Reflexes are normal. No masses or areas of tenderness.

*Extremities.*—The left arm and leg are spastic. He has little power in these members. He can move the arm with slow difficulty. He is unable to move the toes on the left side. The foot can not be dorsally flexed. Biceps and triceps are exaggerated on the left. Prompt on the right.

Patellar reflex on the left markedly exaggerated. Persistent patellar clonus. Ankle clonus also present, but of short duration. Babinski sign is with upward movement of the big toe. Reflexes on the right slightly more than normal.

Station good. Sways and tends to fall to the left though this is probably through weakness, on this side. Gait spastic.

Dysmetria pronounced on the left side. Normal on the right.

Adiadokokinesis present on the left. Slow muscle response, probably due to spasticity. Movement slow but not awkward.

Normal asteriognosis on the left and right. No sensory disturbances in either extremities on either side.

*Summary.*—Convulsions beginning 9 years ago. Feeling of numbness and weakness on the left side before attack. Spasticity in both extremities on the left. More marked in the leg. Reflexes increased on the left. Babinsky and

patellar and ankle clonus on the left. No objective sensory disturbances either side. Headache and vomiting. Yawns frequently.

*Tentative Diagnosis.*—Brain tumor. Right motor area, near Rolandic fissure and high up on the cerebral cortex.

3-10.—*X-Ray Report.*—Pituitary measurements—A. P., 10; depth, 10; sellar measurements same as at previous examination. Normal in size.

Blood and urine findings normal.

3-11.—The patient has great pain in the head. Rolls about and has fallen out of the bed once or twice.

Marked loss of power in triceps on the left and external quadratus on the left and in perineal groups and dorsal flexors of the foot.

Lumbar puncture. Pressure 50 mgs. Hg.

Spinal fluid and blood Wassermann—negative. Bloodtyping—type two.

3-12.—*Eyes.*—Oc. rotations full. Pupils equal, regular and respond to usual reflexes.

O.D.—All margins of disc observed with increased capillarity; measure 4 D. Vessels tortuous. Veins dark and distended.

O.S.—Same picture; disc  $3\frac{1}{2}$  D.

Patient examined about 6 weeks ago on the service of Dr. W. G. Spiller in this hospital. At that time the physical examination was essentially similar to that noted at present. The spinal fluid pressure was only 6 mm. of Hg., the eye-grounds showed only a very early papillœdema, and the headaches were a very much less prominent feature of the case. At that time it was Doctor Spiller's opinion that "the symptoms are those of a progressive hemiplegia of very gradual development, without any stroke associated with Jacksonian paræsthetic attacks on the same side. Condition suggests a lesion of the right parietal lobe extending into the motor area, especially to the centre for the lower limb, either syphilitic or neoplastic, and operation must be considered."

An operation over the right motor and parietal areas was therefore decided upon.

3-14.—*Operative Notes on Craniotomy.*—Findings: Subcortical cyst; evacuation of cyst; injection of air; röntgenogram; routine blood transfusion 500 c.c. blood.

Exploratory craniotomy performed under ether anæsthesia, to uncover the motor cortex. There was evidently tremendous intradural tension for the dura was very tight and did not pulsate. Before opening the dura the ventricle was evacuated by callosal puncture and a quantity of fluid escaped. This relieved pressure somewhat and a dural incision was made exposing the cortex under the lower

half of the opening. The cortex was inspected by reflection of the dural flap from the posterior half of the opening. This was then closed and the anterior half exposed by reflection of the dural flap. There still seemed to be considerable pressure as the brain herniated out through the opening. So great was this pressure that it was thought wise to have a 15 per cent. solution of sodium chloride given intravenously to attempt to dehydrate the brain before closure as there was no tumor present on the cortex. However, before the patient had received 10 c.c. of the salt solution palpation of the cortex revealed an area having a tense cystic feel, a cannula was passed down into this area and about 25-30 c.c. of a clear yellowish fluid escaped. This so relieved the pressure that no more concentrated salt solution was given. Twenty-five c.c. of air was then injected into the cyst, and the dural opening and osteoplastic flap closed. The patient was sent down to the X-ray department for a skiagram. On his return to the ward he was given a routine transfusion of 500 c.c. of citrated blood. Post-operative condition satisfactory.

3-15.—Patient's condition good. X-ray showed large cyst with a filling defect in its wall, apparently not connected with the ventricle.

3-21.—Second stage craniotomy.

Removal of tumor. Probably glioma.

Flap reflected. Cannula introduced into the cyst as at first operation. Withdrawal of straw-colored fluid. Incision  $2\frac{1}{2}$  cm. deep in the cortex, posterior, superior angle of the opening. With a suitable retraction and illumination the cyst cavity was readily seen and presented; at the superior posterior angle a tumor the size of an English walnut, bluish in color, small but encapsulated on the surface. It was readily detached by blunt dissection and removed in its entirety. There was no bleeding from the site of the growth. Its origin could not be determined. It was believed to be the remains of a glioma, the nubbin that one sometimes sees at the terminal process of a cystic degeneration. Hemostasis was completed at the conclusion of the operation. The dural wound and scalp wound were closed. Tubular drainage of skin incision.

3-22.—Patient's condition good. Moving left arm and leg freely. Report from fluid from cyst. Straw colored clear fluid. Cells—occasional lymphocyte. Sugar—plus.

3-28.—The condition of the patient continues to improve. He uses the left hand fairly well and can place it on the head. *Adiadokokinesis* much improved. Stitches out, wound healing rapidly.

Pathological diagnosis: Glioma.

4-2.—Patient up and about ward. He walks with a slightly paraplegic gait and still shows some foot-drop. Wound in good condition. To be discharged after he has received radium treatment.

## DISCUSSION

This case is interesting from two points of view. The symptoms attributable to this tumor were present according to the history for at least nine years. For how many years the tumor was in existence before it became large enough to produce any symptoms is a matter of conjecture. But little is known about the life history of these growths. Gliomata frequently undergo cystic degeneration. Presumably they outgrow their blood supply, tissue death with fluid formation follows, a wall is formed by the inflammatory tissue about the necrotic area and a cyst results. In the cyst wall at some point an area of gliomatous tissue remains which may continue to grow. Tumors of this type are much more adaptable to surgical treatment than are the ordinary infiltrating type of glioma. As in this case, the cyst may be localized and opened, the gliomatous area removed and radium therapy resorted to in an attempt to prevent the cyst from refilling and proliferating. If possible the cyst wall should be removed, but in this case it was not possible to grasp it and strip it away.

At the first operation, once the cyst was found, we were faced by the question as to what the next step should be. One of the difficult decisions to make in cranial surgery is when to stop in a given procedure, even though the immediate condition of the patient is not particularly alarming. The brain tissue responds rapidly to operative insult by swelling. Unless this fact is taken into account and preparation made to deal with this increased intracranial tension it may be found impossible to close the dura and at times the swelling may be so great that the whole bone-flap may have to be sacrificed in order to close the wound. The disastrous results following such a misfortune, adhesions between the unprotected cortex and the skin flap, the presence of a large bony defect in the skull, would prevent the patient from enjoying the full measure of relief to be expected from the removal of a tumor. The replacing of the fluid contents of a cyst with air is a simple procedure. It enables the operator to obtain a clear conception of the problem with which he has to deal, it assures him that the fluid he obtained came not from the ventricle, but from a cyst. The dimensions of the cyst are clearly defined, and in some cases, as in this, the nubbin of actively proliferating tumor

lying in the cyst wall may be seen with enough distinctness to make its location certain and its removal much more simple. While we do not advocate dividing all cranial operations into two stages, yet we do not hesitate to do so if occasion demands. It is much better to do too little at one sitting and have the patient recover, then re-operate with certain knowledge of the conditions to be faced, than it is to do a brilliant extirpation of a tumor with the patient dying of the shock of the operation, or if he lives facing the necessity of a second procedure to repair his cranial defect, or more difficult still, to remove the adhesions which may be crippling the function of important centres in his cerebral cortex. We claim no originality in this suggestion. Horrax<sup>1</sup> reports a similar case from Cushing's clinic and doubtless a close examination of the literature would reveal others. It seems to us, however, to be a very safe and common-sense way of dealing with an intracranial condition which if inexpertly handled may involve both operator and patient in disaster.

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<sup>1</sup> "Archives of Neurology and Psychiatry," June, 1921, Vol. v, pp. 7-9, *Trans. of Boston Soc. of Psychiatry and Neurology*.

## RHINO-LARYNGOLOGIC PHASES OF HARELIP AND CLEFT PALATE WORK \*

BY WARREN B. DAVIS, M.D.

PROBLEMS of rhinologic interest and importance are presented by all harelip and cleft palate cases, since part of the deformity in each case is in the degree of deviation of the nasal septum associated with widening and flattening of the nostril and a corresponding malposition of the ala nasi. In some cases the nasal septum is elongated. In rarer instances the septum is rudimentary or may be absent. Laryngologic phases are presented by every cleft palate case, since the selection of the operative measures which will give the nearest perfect functional result in the palate muscles is of the greatest importance in securing improved phonation, articulation and quality of voice.

A brief review of the embryology of the nasal and oral areas may aid in more clearly understanding the formation of the various types of these congenital deformities or arrested developments. During the third week of embryonic life there is evidence of beginning development of the nasal area as shown by increasing thickness of the ectoderm on the anterolateral portions of the forebrain. At the end of the third week, or during the fourth week, the nasal area appears as a depression, which is brought about by the increased thickness of the surrounding mesenchyme. The primitive nasal capsule develops as a part of the primordial cranium. From that part which extends forward beyond the anterior portion of the notochord a core is formed for the frontonasal process—a relatively broad mass of tissue separating the nasal pits—and from its antero-inferior portion the premaxilla and the philtrum develop. On each side are the lateral nasal and the maxillary processes. By the approximation of these processes with the median nasal process, their ectodermal coverings are brought into contact. Normally the interposed ectoderm is soon absorbed, the processes become united by mesoderm, thus forming the upper lip and the floor of the primitive anterior

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\* Paper read before the Section on Otology and Laryngology of the College of Physicians of Philadelphia, March 15, 1922.

nares. The maxillæ develop in the connective tissue of the maxillary processes, on the medial sides of which the palatal ridges or shelves appear from the forty-fifth to the forty-eighth day of embryonic life. By the sixtieth to the sixty-fifth day these processes normally approximate and the palate is formed—the palatal processes uniting not only with each other, but also with the premaxilla and with the lower margins of the median nasal process, in which the vomer has its origin (Figs. 1 and 2).

Union between the processes normally takes place from before backward. A failure of union between the median and the lateral nasal processes and the maxillary process produces a complete harelip. Failure of union between the maxillary process and the premaxilla produces a cleft in the alveolar process. Want of union between the palatal shelves produces cleft palate. Any of these conditions may be partial or complete, unilateral or bilateral.

Since 1915 Dr. J. Chalmers DaCosta has very kindly given me the privilege of operating upon the harelip and cleft palate cases admitted on his service at the Jefferson Hospital. These cases, plus my private ones, form a series of one hundred and fifteen. Some of the varieties of these congenital defects are illustrated here by the photographic records of cases selected from the combined series.

*Median harelip* is very rare and is due to agenesis of the philtrum. It is usually associated with failure of development of the premaxilla and other structures having their origin from the median frontonasal process. Such a condition is shown in Case I (Fig. 3), age four months, weight five pounds. Referred to Doctor DaCosta's service by Dr. Arthur Wagers. The philtrum, premaxilla and the nasal cartilages were wholly wanting. Posterior to the region normally occupied by the premaxilla the palate was well formed (Fig. 4). The nasal bones were very rudimentary and produced no nasal elevation. The nasal septum was entirely absent anteriorly and was markedly deficient posteriorly—being represented only by a *very* short mesethmoid plate which had no inferior attachment. The vomer was exceedingly rudimentary—not extending far enough antero-inferiorly to come in contact with the palate. Thus there was a single nasal cavity only partially subdivided in the extreme postero-superior portion by the very short mesethmoid. The medial

surfaces of the inferior and middle turbinates were rather closely approximated and oftentimes in contact. These conditions were demonstrable by direct inspection and also by the X-ray examinations which were made by Dr. W. F. Manges.

The child's condition was very poor when admitted to the hospital and was never gotten into a condition which would permit operation. Enterocolitis developed and resulted in death. Had the general condition permitted operation, we expected to repair the cleft in the lip and to construct a better margin for the single nostril, leaving any further operative procedures until a later period of development.

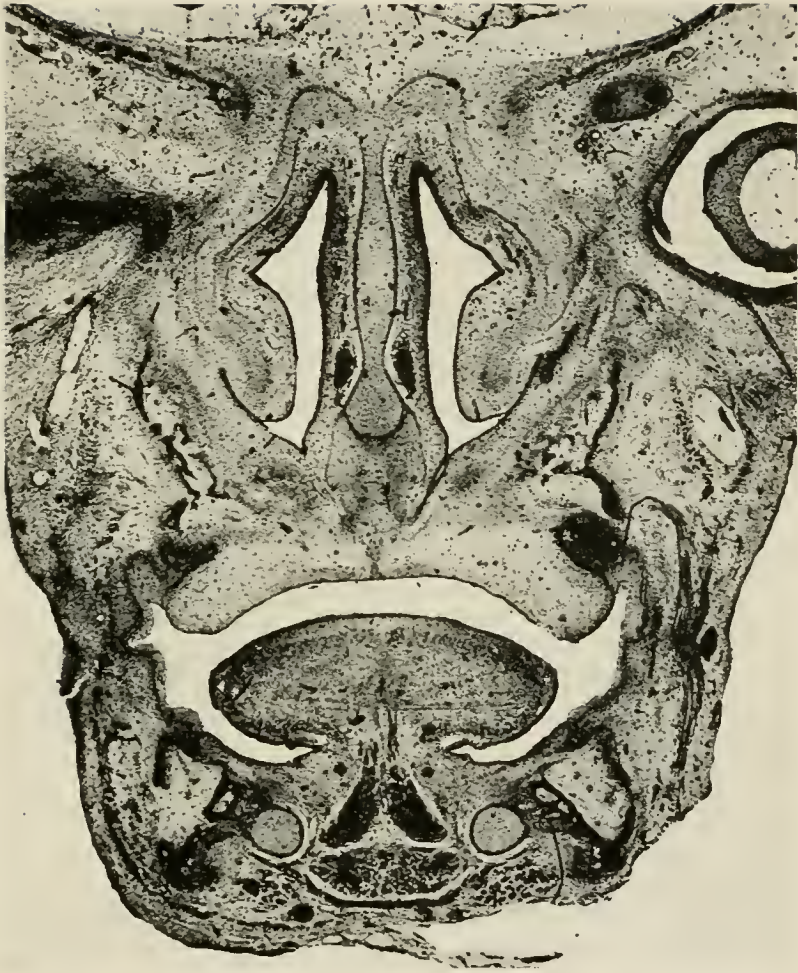
Besides the morphologic abnormalities shown in the nasal and oral areas of this case, other stigma of degeneracy showed the child to have been an idiot of the Mongolian type. The Chinese-like appearance was very striking, as was also the unusual pigmentation of the skin. A Wassermann examination of the spinal fluid, taken immediately after death, was negative.

The mother was apparently a normal American, nineteen years of age. The father, twenty-four years of age, was of German parentage. They had one other child, born about eighteen months before the above described case, which had complete double harelip and cleft palate, said to have been of such an unsightly type that the mother was never allowed to see the child, which lived only eleven days. No history of other such defects could be obtained on either side of the family.

*Unilateral Harelip.*—Cases of single, simple harelip, complete or incomplete, show varying degrees of widening of the nostril with flattening of the ala nasi on the affected side, and deviation of the anterior portion of the nasal septum toward the opposite side (Figs. 5 and 7). In the vast majority of incomplete cases there is either deficiency or absence of muscle tissue between the upper angle of the cleft and the floor of the nostril. To thoroughly correct such deformities it is necessary to convert the incomplete clefts into complete ones in order to secure proper approximation of muscles and to correct the flattening of the ala nasi and the accompanying wideness of the nostril.

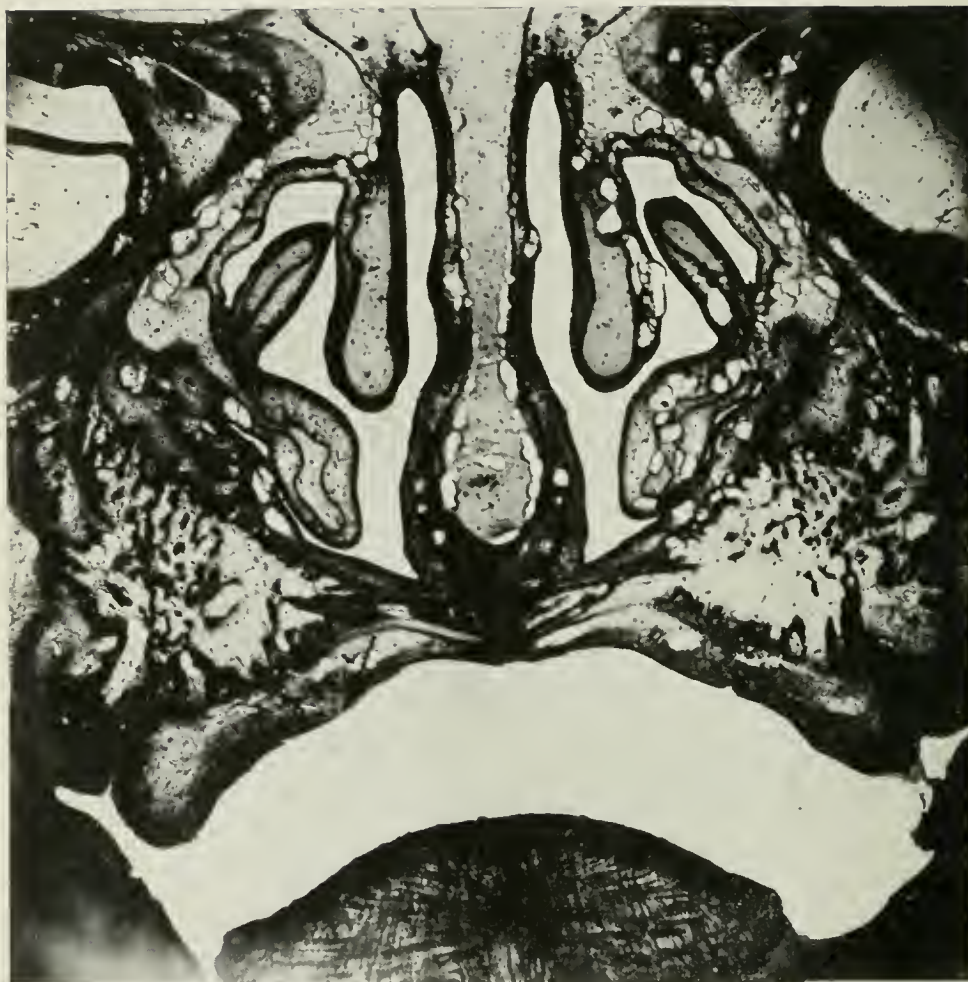
In outlining incisions for the correction of harelip deformities, the method devised by J. E. Thompson (Fig. 8) has been decidedly

FIG. 1.



Microphotograph of a coronal section through the oral and nasal areas of a sixty-day embryo, just posterior to the premaxilla. (Series A, No. I, slide 9, section II.) Showing fusion of maxillary processes with each other and with the nasal septum.

FIG. 2.



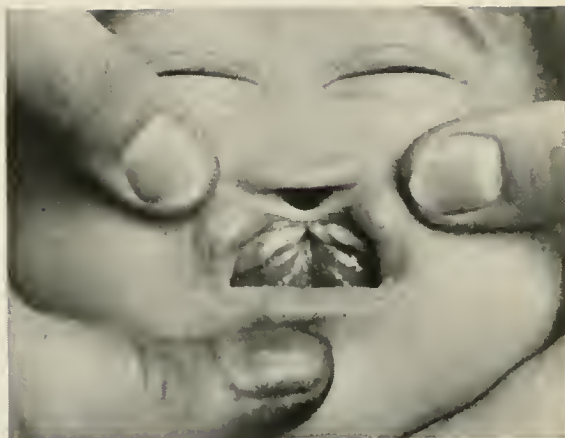
Microphotograph of a coronal section through the maxillary and nasal areas of a ninety-five-day embryo, showing extent of ossification extending into horizontal processes of maxillæ. (Series A, No. 6, slide 10, section 1.)

FIG. 3.



Case I.—Age four months. Median harelip. Premaxilla, philtrum and nasal cartilage wholly wanting. Nasal bones, mesethmoid and vomer are rudimentary. Mongolian idiocy.

FIG. 4.



Case I.—Showing the single central nostril and the well-formed palate posterior to the area normally occupied by the premaxilla.

FIG. 5.



Case II.—Age five months. Incomplete unilateral harelip, showing scantiness of muscle tissue in the upper portion of lip between the angle of the cleft and the floor of the nostril, with consequent widening of the nostril, flattening of the ala nasi and moderate deviation of the nasal septum.

FIG. 6.



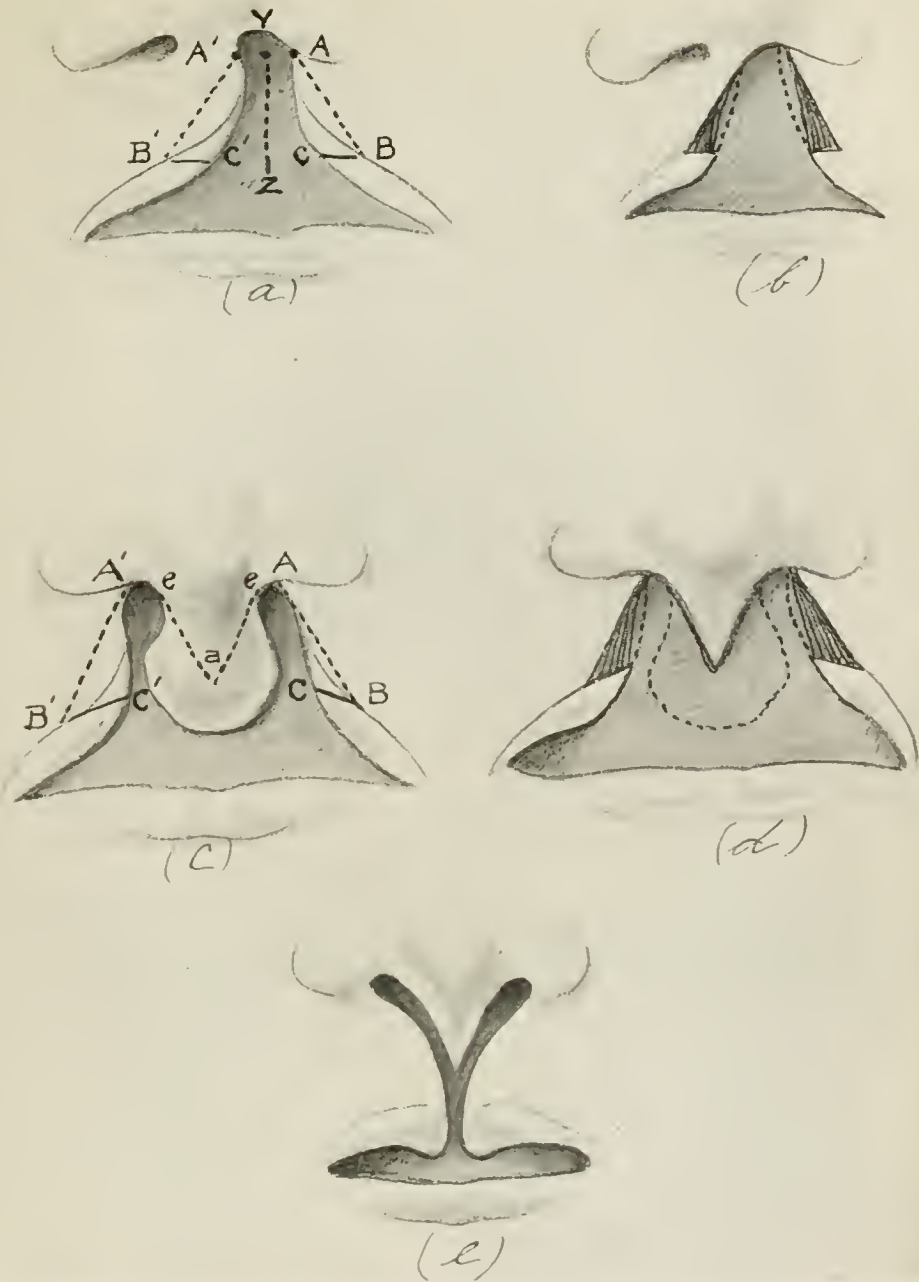
Case II.—Showing contour of lip and position of the ala nasi eleven weeks after operation.

FIG. 7.



Case III.—Age three weeks, showing complete unilateral harelip, with marked flattening of ala nasi.

FIG. 8.



Semi-diagrammatic sketches of Case III and Case VIII showing lines of incisions used for the correction of single and double harelip. (After the methods of J. E. Thompson.) In single clefts (a) sharp pointed calipers are used in measuring the distance (YZ) from the mid-point of the floor of the nostril to the point in the same sagittal plane to which the free margin of the lip would come if it were of normal contour. Fixing the distance on the calipers and keeping the superior point at Y, the inferior point of the calipers is rotated describing an arc which crosses the vermillion border of the lip on each side of the cleft. These points B and B' are distinctly marked by making a puncture with the point of the calipers or with a small scalpel. Point C and C' are then located on the free margin of the lip so that the angles ABC and A'B'C' are between 70 and 80 degrees. Incisions carried through the entire thickness of the lip with a small scalpel at a right angle to the skin surface and following the lines as outlined will give surfaces for approximation which are of equal length and which, when sutured together, will give a lip the length of which is the estimated normal length (YZ) plus the distance from the vermillion border to the free edge of lip (CB) which is usually just sufficient to allow for subsequent contraction. Sketch b shows tissue removed. Lines of incision used in double harelip are shown in sketch c. The philtrum is trimmed to a V-shape, leaving as much tissue as is possible with thorough removal of the vermillion borders. The lateral incision lines are outlined as described for single harelip. Sketches d and e show tissues ready for approximation.

FIG. 9.



Case IV.—Age five months. Complete unilateral harelip and cleft palate, showing marked flattening of ala nasi and deviation of nasal septum.

FIG. 10.



Case IV.—Twenty-two months after first operation, showing position of alveolar process and teeth.

FIG. 11.



Case IV.—Showing contour of lip and nostrils twenty-five months after first operation.

FIG. 12.



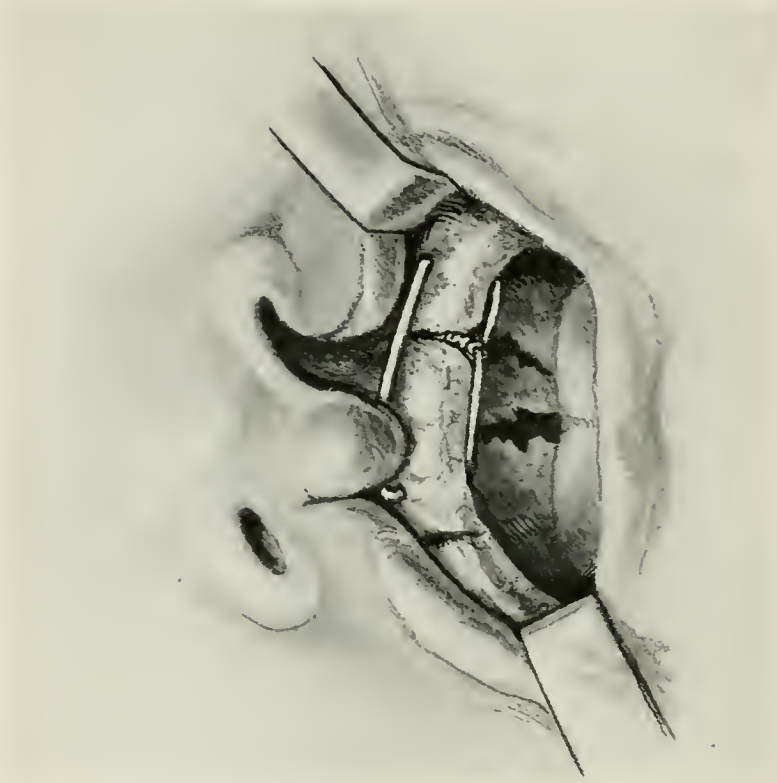
Case V.—Age nine days. Complete unilateral cleft palate, incomplete harelip.

FIG. 13.



CASE V.—Showing contour of lip and nostril six months after operation. The excess fulness at the lip margin will be corrected at the same time the posterior portion of cleft in palate is closed.

FIG. 14.



Sketch showing method of approximation of margins of alveolar portion of cleft, and the position of silver wire holding premaxilla in position.

the most satisfactory in our cases—both at the time of operation and in the end results. Before approximating the margins of the cleft it is best to free the superolateral portions of the lip from the anterior surfaces of the maxillæ through incisions made above the alveolar process. This not only relieves tension but permits the ala nasi to be brought into proper position at the floor of the nostril. The nasal septum is forced into better position by the use of a Sinexon nasal dilator.

*Unilateral Harelip and Complete Single Cleft Palate.*—These cases, as compared with the preceding group, show wider separation of the lip margins, more marked flattening of the ala nasi, varying degrees of antero-superior rotation of the premaxilla, and greater deviation of the nasal septum (Figs. 9 and 12). Correction of the deformities is preferably done by a two-stage operation, closing the alveolar cleft and repairing the harelip at the first operation, the cleft in the palate being repaired a few months later. We prefer to do the first operation as soon as it is determined that a suitable feeding formula has been selected and the child shows a steady gain in weight. This time varies from the second week to the third month. The second operation is preferably done sometime between the twelfth and the twentieth month, always selecting a time when the child's general condition is good.

If the alveolar cleft is narrow, or if the patient is quite young, the margins of the alveolar cleft can usually be approximated by digital pressure supplemented by pressure against the lower portion of the nasal septum and the floor of the nostril by means of a Sinexon nasal dilator. This brings the premaxilla into approximately the normal position, and with it the nasal septum is brought to the median line. In older cases, or when the separation is very wide in infants, it may be necessary to partially divide the alveolar process on its buccal surface just posterior to the canine region. This allows a green-stick fracture at that point when the premaxilla is brought into position. The margins of the alveolar cleft are held in apposition by a silver wire applied as shown in Fig. 14. The correction of the anterior bony deformity brings the margins of the cleft in the lip much closer together, after which the lip repair is essentially the same as described in simple harelip. Fig. 10 shows

the contour of the alveolar process twenty-two months after operation. The expectation is that but slight orthodontic work will be required in such a case, presuming that the permanent teeth will be in as good position as the deciduous ones.

In closing the cleft in the palate we usually use a modified Langenbeck muco-periosteal flap-sliding method, since we obtain by that procedure the best functioning palatal muscles. Incisions are made on each side just medial to and parallel with the posterior portion of the alveolar process. Muco-periosteal flaps are loosened from the partial bony palate, then separated from the nasal mucosa by dividing the attachment of the nasal mucosa along the posterior edges of the palate bones. When the width of the cleft anteriorly is such as to make additional width of flap necessary, the muco-periosteal flap dissection in single cleft cases can be carried around the lower margin on the attached side and  $\frac{1}{5}$  to  $\frac{1}{4}$  inch of mucosa taken from the lower portion of the nasal septum. The best possible blood supply is preserved, but the flaps must be without undue tension when approximated. This may necessitate further extension of the original incisions posteriorly.

Cases in which all operative procedures are completed before the end of the second year give better articulate speech and nearer normal voice quality than do those completed at a later period. For this we believe there are two reasons: First, the individual does not have to be trained out of old habits of cleft palate speech, and second, the muscles of the palate usually develop to a greater extent when repaired early than they do when the cleft is allowed to persist till later childhood or adolescence. This gives not only better muscle control, but the increased antero-posterior extent of the soft palate permits better closing off of the postnasal space, and consequently there is less of the nasal quality of voice.

After operative procedures have been completed we always advise that the patient be given training by one familiar with the best methods for the correction of speech defects. The results obtained in our cases taught by Mrs. M. S. Steele have been very gratifying. Articulation is always improved by the training. The ultimate extent to which nasal sounds can be decreased and the ability to direct sounds through the mouth increased under such training depends

FIG. 15.



Case VI.—Age 22. Showing result of operation for double cleft palate (clefts extended to premaxilla), one year after operation. Palate muscles are shown held tense.

FIG. 16.



Case VI.—With palate muscles relaxed. Note good contour and length of soft palate.

FIG. 17.



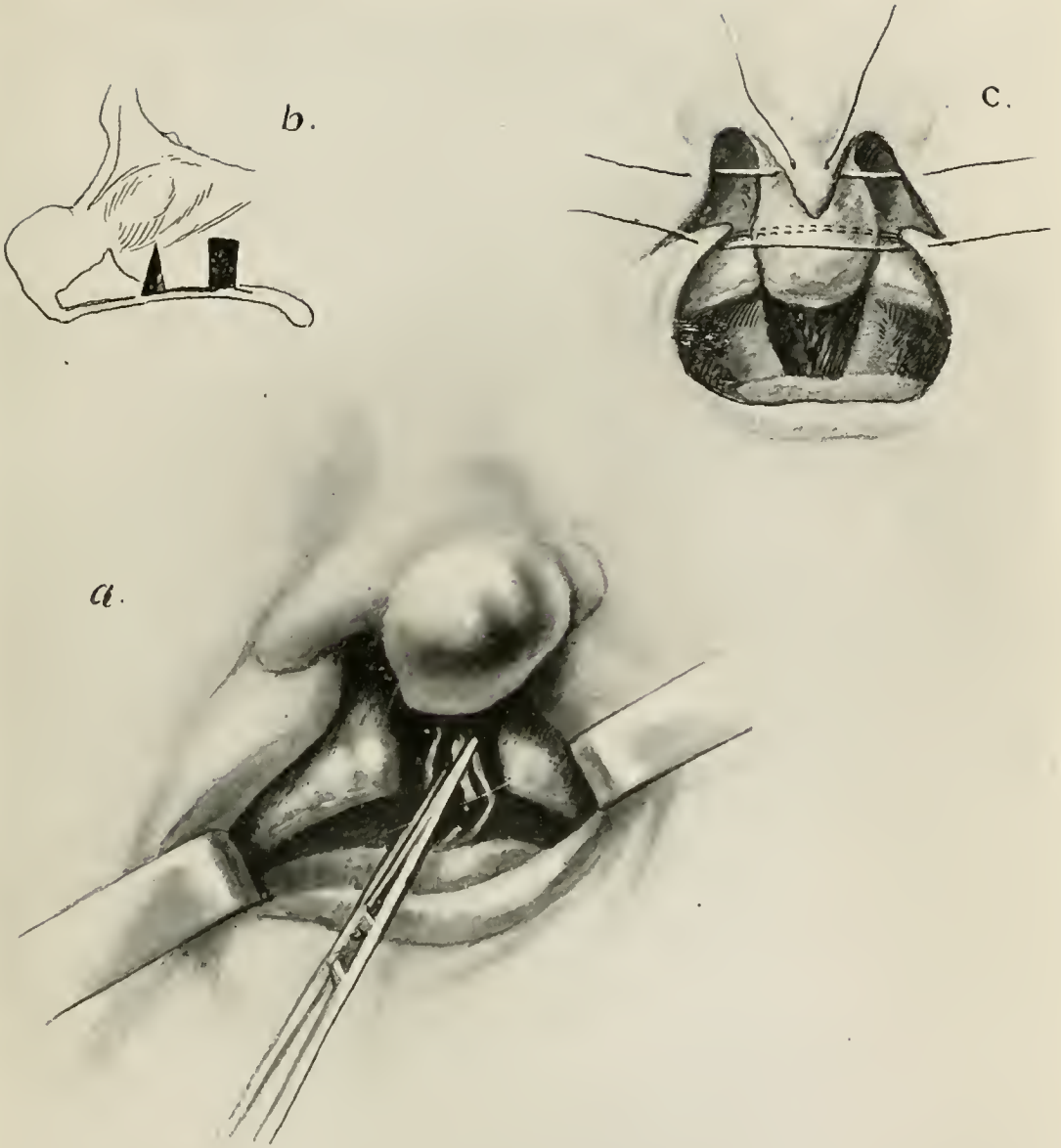
Case VII.—Showing result of operation for complete harelip and cleft palate on left side and incomplete cleft palate on right side, after patient was twenty-one years old. The degree of shortness of the soft palate precludes the possibility of developing a speaking voice of good quality.

FIG. 18.



Case VIII.—Age two months, showing complete double harelip and cleft palate with marked elongation of vomer and antero-superior rotation of premaxilla. Note absence of columella, the philtrum extending directly anteriorly from the tip of the nose.

FIG. 19.



Semi-diagrammatic sketches of operation on Case VIII. Sketch *a* shows the submucous resection of the triangular and rectangular sections from the vomer and the nasal cartilage to allow the premaxilla to be rotated and placed in normal position to complete alveolar arch. The antero-posterior extent of the sections removed (sketch *b*) varies in direct proportion to the extent of rotation and elongation to be corrected. In most cases the triangular section alone is sufficient. Sketch *c* shows premaxilla in position and lip ready for suturing.

FIG. 20.



Case IX.—Age fourteen days. Complete double harelip and cleft palate, with premaxilla rotated laterally and nasal septum markedly deviated.

FIG. 21.



Case IX.—Posterior view of anterior section, showing union in lip, marked deviation of nasal septum and rotation of premaxilla.

upon the length of the palate, the degree of muscle control and freedom from undue tension by fibrous contraction.

CASE VI (Figs. 15 and 16) had a wide double cleft palate extending to the premaxilla, which was operated upon at the age of twenty-three years. Notwithstanding the long postponement of operation the muscles of the soft palate were unusually well developed, permitting an operative result in which the palate has sufficient length to close off the postnasal space and consequently a most gratifying improvement, both in the articulation and in the quality of the voice.

CASE VII (Fig. 17) had a complete harelip and cleft palate on the left side and partial cleft palate on the right side. Parental objection to operations caused postponement of surgical procedures until after the twenty-first year. The palate muscles were poorly developed, thus giving an operative result in which the palate is so short that it is impossible, by any amount of training, to close off the postnasal space. Training has greatly improved the articulation, but the quality of the voice must necessarily retain much of the nasal tone.

*Bilateral Harelip and Complete Bilateral Cleft Palate.*—Complete bilateral clefts present varying degrees of antero-superior rotation of the premaxilla, elongation of the vomer and nasal cartilage and shortening of the columella and philtrum (Fig. 18). In some instances there is in addition a lateral rotation of the premaxilla and an associated deviation of the septum (Figs. 20 and 22).

If the antero-superior rotation of the premaxilla is marked, it is advisable to remove submucously a triangular section of the anterior portion of the vomer and nasal cartilage of just sufficient size to permit infero-posterior rotation of the premaxilla to its normal position. The base of the triangle removed should be equal to the arc through which the premaxilla is to be rotated. The incision for the submucous removal is made in the midline on the lower margin of the vomer just posterior to the premaxilla (Fig. 19). The mucosa is then removed from the sides of the premaxilla and from the antero-medial margins of the alveolar process on each side so that raw surfaces are brought into apposition when the premaxilla is rotated into position, and medial pressure is made upon the alveolar processes. A silver wire through the alveolar processes and in front of

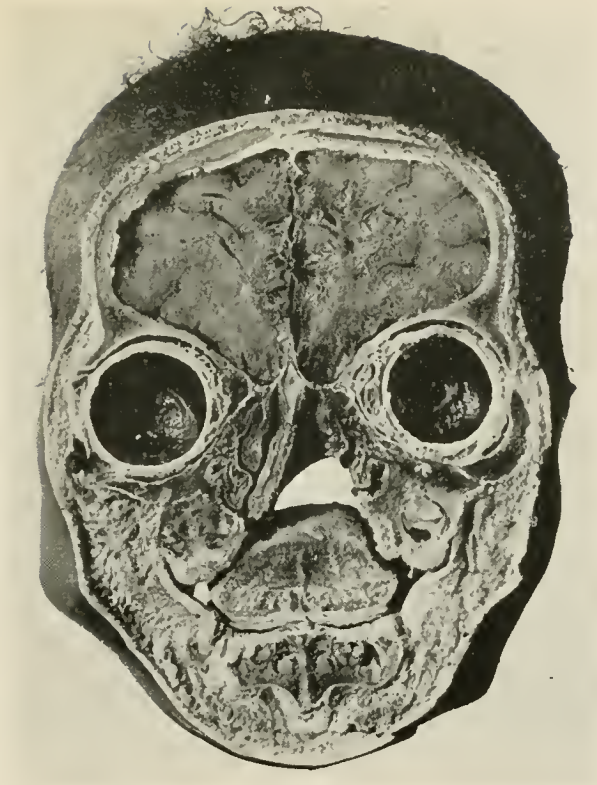
the premaxilla, as indicated in Fig. 19 c, holds the surfaces in contact and the premaxilla in position. The philtrum is trimmed to a "V" shape just within the vermilion borders. Measurements for the incisions in the lip are made by the Thompson method (Figs. 8 and 19). The lip must be freely separated from the anterolateral surfaces of the maxillæ to allow proper formation of the nostrils. The highest sutures in the floor of the nostrils must be carefully placed to bring the alæ into proper relation.

CASE IX (Fig. 20) is unusual in the degree of lateral rotation of the premaxilla, in the marked deviation of the nasal septum (Fig. 21) and in the width of the cleft in the palate. Moreover, harelip and cleft palate defects are very rare in the negro race, this being the only such case we have seen. The double harelip was repaired on the fourteenth day and the alveolar margins partially approximated by lateral pressure and held by silver wire suture. The cleft was too wide for immediate complete approximation. Good union was secured in the lip and a rather good African type of contour was obtained in the alæ of the nostrils. We hoped to further approximate the margins of the alveolar cleft by a several-stage operation. Intestinal and feeding troubles developed, however, and in spite of the good care and attention given by the Pediatric Department, death resulted four weeks after operation. The sections of the head from which Figs. 21 and 22 were photographed are now in the Museum of the Jefferson Medical College.

*Bilateral harelip and bilateral cleft through alveolar process*, with antero-superior rotation of premaxilla, but with a well-formed palate posterior to the region normally occupied by the premaxilla (Figs. 26 and 28). This type of malformation is uncommon. The vomer, as shown in Fig. 28, was markedly elongated and was not attached to the horizontal processes of the maxillæ in their anterior portions, thus allowing the vomer and the attached premaxilla to be easily moved from side to side.

The deformity was corrected by a single operation when the child was four weeks old. The premaxilla was brought into normal position by the same procedure described in Case VIII. The lateral surfaces of the premaxilla were removed down to the periosteum and the mucosa was removed from the margins of the alveolar process

FIG. 22.



Case IX.—Anterior view of second section, showing the septal deviation, the great width of the left in the palate and the rudimentary horizontal processes of the maxillæ.

FIG. 23.



Case X.—Age four weeks. Double hare-lip and cleft palate, incomplete on left side.

FIG. 24.



Case X.—Showing form of lip and nostrils nine days after operation.

FIG. 25.



Case X.—Showing contour of nostrils two months after operation.

FIG. 26.



Case XI.—Age six weeks. Bilateral harelip and bilateral clefts in alveolar process, but not extending into the horizontal portion of palate.

FIG. 27.



Case XI.—Showing antero-superior rotation of premaxilla and the shortness of the columella.

FIG. 28.



Case XI.—Showing the well-formed palate posterior to the area normally occupied by the premaxilla.

FIG. 29.



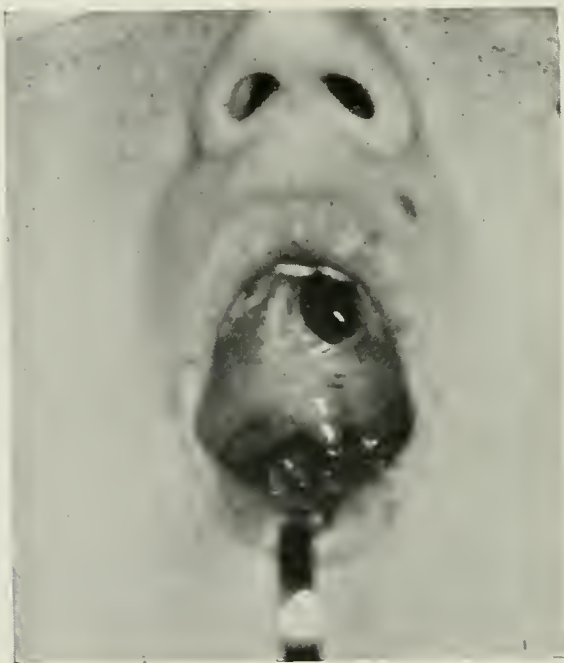
Case XI.—Showing contour of lip and nostrils sixteen days after operation.

FIG. 30.



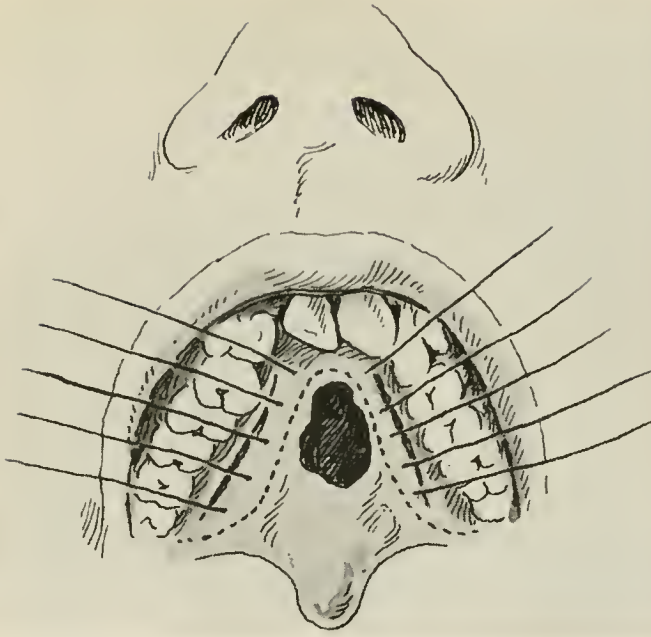
Case XII.—Age seventeen years. Opening in hard palate after an operation for double cleft palate, done when the patient was one year old.

FIG. 31.



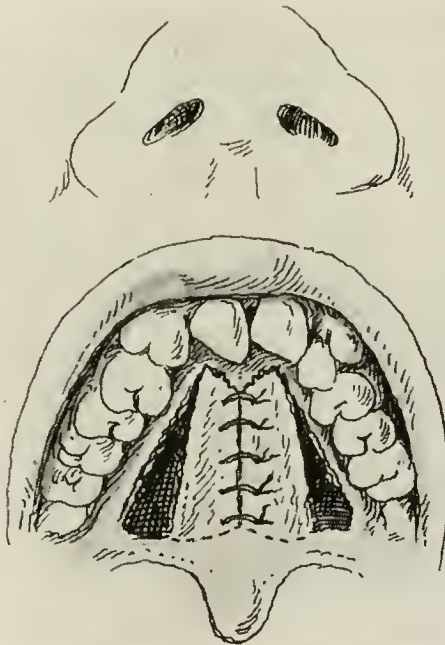
Case XII.—Showing good union of the soft palate and the opening in the hard palate.

FIG. 32



Case XII.—Semi-diagrammatic sketch. Dotted line indicates margins of bony cleft. Lateral incisions are shown, through which the bones were divided.

FIG. 33



Case XII.—Semi-diagrammatic sketch showing approximation of the margins of the opening in the hard palate. The site of the green-stick fracture produced anteriorly is indicated, as are also the posterior ends of the bones in their new position.

FIG. 34.



Case XII.—Photograph of palate two months after operation.

FIG. 35.



Case XII.—Photograph of posterior portion of palate, two months after operation.

and along the anterior edge of the horizontal portion of the palate. The mucosa on the posterior surface of the premaxilla along the line coming in contact with the horizontal portion of the palate was also removed, thus obtaining raw surfaces for all points of approximation. The premaxilla fitted snugly into position and was held by a silver wire. The double harelip was repaired as shown in Fig. 8. Complete union was obtained throughout. Fig. 29 shows the form of nostrils, tip of nose and lip, sixteen days after operation. The shortness of the columella caused marked depression of the tip of nose at the time of operation. Anatomic approximation of the parts, however, permits normal function of muscles which continually aids in improving the condition, as is shown by the rapidly increasing length of columella and philtrum.

*Incomplete Union in Hard Palate.*—Secondary repair of incomplete union in the palate is often more difficult than a primary operation, since there is less tissue for flap formation, poorer circulation and varying amounts of scar tissue. Figs. 30 and 31 show a type not infrequently encountered. Such an opening precludes the possibility of good articulate speech. This type of defect may be corrected by one of three methods.

1. By making lateral incisions just within the alveolar process on each side, loosening muco-periosteal flaps and approximating them in midline—similar to the method used in the Langenbeck operation for primary closure.

2. By turning a muco-periosteal flap from one side—keeping its attachment at the margin of the opening and carrying the free edge beneath a double-pedicled flap on the opposite side and suturing it there, as by the Lane method.

3. By bringing part of the horizontal processes of the maxillæ and palate bones medially with the attached soft tissues.

We most frequently use the first method in closing such openings. The second is sometimes preferable when the opening is just posterior to or is lateral to the premaxilla. The third method is essentially the plan originally devised by Ferguson, advocated in Philadelphia for many years by J. Ewing Mears and later revived, improved and most successfully used by W. J. Roe in his primary cleft palate operations.

The case shown in Figs. 30 and 31 had been operated upon in another city when she was one year old. She is now seventeen. The extent and position of the horizontal processes of the maxillæ and palate bones were such that we decided the third method was applicable. The mucous membrane was removed from the margins of the opening, carrying the incisions a short distance both anteriorly and posteriorly beyond the margins of the opening making its outline somewhat elliptical. Lateral incisions were made as shown in Fig. 32. The horizontal processes of the maxillæ and the palate bones were cut through along these lines and the overlying nasal mucosa also divided. Forcing the strips of bone and the attached soft tissues medially produced a green-stick fracture at the anterior attachments of the bones. Small holes were made in the partially detached bones and the soft tissue covering them, through which interrupted 00 wire sutures were passed and tightened sufficiently to hold the medial margins in apposition, as shown in Fig. 33.

An advantage of this method in this case was that the change in the position of the bones increased to an appreciable extent the length of the palate. Union was complete. The lateral openings closed by granulation in sixteen days. Figs. 34 and 35 show the condition of the palate two months after operation. The improvement both in articulate speech and in the quality of voice has been rapid and exceedingly gratifying.

## SOME POINTS OF THE CLINICAL PATHOLOGY OF CANCER OF THE RECTUM

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CURLING said that cancer of the rectum was usually seated at seven centimetres from the anus, but any portion of the rectal surface may be the seat of this process. According to their site it is easy to classify malignant growths of the rectum in high, medium and low cancer. This classification is classic. It is the one that distinguishes ampullar, supra and infra-ampullar cancer. Quénu and Hartmann have described four types of rectal cancer to which they accord the following limits:

(1) A low-seated cancer of the anus arising below the insertions of the levator ani in the transverse sphincter of the anus.

(2) A medium infra-peritoneal cancer developing in the rectal segment comprised between the levator ani and peritoneum, otherwise put, ampullar cancer.

(3) A high-seated cancer having its lower limits at the level of the peritoneal cul-de-sac and extending as far as to reach or to pass beyond the origin of the omega loop.

(4) A type of cancer totally invading the rectum, represented by those cases in which this segment of the bowel is involved from the anus to twelve or fifteen centimetres above it.

All writers are careful to state that classifications suffer great and many exceptions, and in point of fact a rectal cancer may easily exceed the limits adopted.

Total rectal cancer is rare and it is exceptional that the recto-sigmoid junction, ampulla and rectal canal are all involved in the malignant process. Cancer of the rectum is more prone to extend outside the organ than into its walls having—contrary to what takes place in gastric cancer—a rapidly extrinsic extension. It is true that out of a total of twenty-one specimens, Quénu and Hartmann found five in which the cancer was total, but foreign writers seem to have rarely met with it.

Braïtsef only found one total rectal cancer out of twenty-seven

specimens and out of a total of thirty specimens that I have examined there was not one. On the other hand, it does not seem to me that the peritoneal cul-de-sac can serve to establish a topographical limit because clinically it is an inappreciable landmark and above all, as I shall show farther on, it is frequently involved.

Besides, is it possible to choose as characteristic the invasion of the sphincter by a low-seated rectal cancer? The clinical signs of this invasion are occasionally of striking distinctness, it is true. For example, in a case recorded by Thiroloix and Mora, the sphincter-algia almost appeared to suddenly announce the irruption of the neoplasm into the sphincter, but frequently digital examination will not decide the surgeon as to the integrity or involvement of this muscle. Finally there are cases of anal cancer in which the sphincter can be partially preserved at operation and afterwards fulfills its important physiological part as the following case shows.

A male, *æt.* 60 years, presented an ulcer with raised edges at the level of the anus encroaching upon the skin and mucosa. It was the size of a one dollar silver piece. No inguinal lymph-nodes could be detected.

The ulcer was freely excised by Lecène without completely dividing and excising the sphincter. Wound closed by deep and superficial catgut sutures; recovery simple with perfect continence. Histologically the lesion was a baso-cellular epithelioma.

The patient was seen one year afterwards without any trace of recurrence and the functions of defecation were normal. He died a few months later of angina pectoris.

Keeping only in mind the three principal sites of rectal cancer, it is interesting to estimate their relative frequency. I have examined this question by a study of twenty-two specimens rather than by the clinical data belonging to the patient. Using the following limits, *viz.*, from the anal margin to about four centimetres above (low-seated cancer), from four to ten centimetres above the anus (mid-seated cancer), above ten centimetres above (high-seated cancer), I found:

Low-seated cancer	.....	2
Mid-seated cancer	.....	14
High-seated cancer	.....	6

Of the twenty-one cases studied by Quénu and Hartmann, they found:

Low-seated cancer .....	5 = 23%
Mid-seated cancer .....	9 = 43%
High-seated cancer .....	2 = 14%
Rectum totally involved .....	5 = 23%

I have looked up the findings of other surgeons in respect to this question and have added the statistics of Kocher (74 cases), Funke (236 cases), Zinner (164 cases) and Bräitsef (28 cases). The total added to Quénu and Hartmann's statistics gives the following figures:

Low-seated cancer .....	51
Mid-seated cancer .....	348
High-seated cancer .....	130
Rectum totally involved .....	6

Consequently, out of a total of 524 cases, cancer of the rectal ampulla constitutes nearly 66 per cent. of rectal cancer, cancer above the ampulla 25 per cent., low-seated or ano-rectal cancer 10 per cent. and total rectal cancer, 1 per cent.

My own statistics are perfectly in accord with these figures. They show that the average distance of the cancer from the anus is 8.2 centimetres.

An attempt has been made to discover upon which aspect of the rectum malignant growths occur most frequently and most observers agree that it is the anterior aspect. Zinner, having found that this site occurred in 75 per cent. of the cases, thought that the explanation could be found in a mechanical irritation from the feces which is far more severe on the anterior than on the posterior surface of the rectum. The latter is more easily depressed; on the contrary the anterior rectal surface lies on the uterus or prostate.

If the frequency of recto-sigmoid cancer appears greater than statistics show it is because operations are at present much more extensive, hence the study of the specimens removed is more complete and easier as well. In this respect it is to be recalled that James Gantlie, after numerous sigmoidoscopic examinations, has reached the conclusion that the point of junction of the rectum with the sigmoid flexure is marked by a stenosis which he proposes to designate

by the name of *sigmo-rectal pylorus*. Hence out of all rectal cancers 25 per cent. of them are seated in the sigmo-rectal pylorus.

*Shape and Aspect.*—In his classic treatise on diseases of the rectum, Mollière says: “Annular, nodular, *en plaque* and gimlet cancers have been described, but no matter how fertile a writer’s imagination may be, observers have never been able to conceive all the types that nature has created.”

The variety of the descriptions, the multiplicity of comparisons, and classifications are most inadequate in fact, when they are compared to the variety of aspects that rectal cancer can assume, from the small, indurated crateriform bleeding ulcer, from narrow annular stenosis up to voluminous budding growths with a more or less large area of implantation. On the other hand how many are the cases that can be easily classified.

The old English classifications of Allingham, Cripps and others, describe a tuberos form, a lamellar form (infiltrating growth) and an annular form.

Quénu and Hartmann, reverting to Trelat and Delens’ classification, propose to make a simpler distinction, namely, a circumscribed and a diffuse form, the latter being able to manifest itself as such from the onset and “then take on a very rapid evolution.”

This classification has been accepted in some recent text-books, but although the study of cancer at its onset is surgically the most important, should this classification be accepted? Perhaps rectoscopic examinations will show that at the onset of their evolution cancers are either deep and infiltrated for a long time, or superficial and ulcerating early and which later on will become indifferently circular, stenosing, circumscribed or budding, diffuse or massive.

Contrary to the more recent text-books, I believe that annular cancer should be distinctly individualized, because although it may be the terminal phase of the other forms and although it may offer a very long evolution, it may, on the other hand, exist at a very early phase of the process and during many repeated examinations it may present both clinically and anatomically a very peculiar physiognomy, just as it has its own peculiar symptomatology and complications.

It appears to me that there is less need of from the start distin-

guishing between a circumscribed and a diffuse form. Evidently degrees in the extent of the lesion exist as I shall show, and if by clinical examination one cannot exactly outline the limits of the growth, it is perfectly true also that there are few instances where, on the autopsy table, the neoplasm appeared to be total and massive, while there are still fewer specimens obtained from operation which have presented this absolute diffusion of the neoplastic process.

Then again, this massive extension of cancer is, in all events, almost invariably secondary and acquired; "the sudden explosion" of a cancer into the entire rectum is an exceptional process. There are many more cancers becoming rapidly annular, than cancers precociously massive; the former require surgical treatment in haste, the latter will always discourage the surgeon.

While in cancer of the stomach and from the viewpoint of operative diagnosis its external shape is most important to ascertain in the case of rectal cancer its internal mucous aspect is more accessible. By the proctoscope one can distinguish between an ulcerated, vegetating or infiltrating neoplasm.

By digital examination it is often difficult to distinguish between a budding ulceration, an irregular tumor and an amfractuous stricture. It is only by an examination of the specimens that one has been able to describe three forms, *viz.*, cancerous tumor, ulceration and stenosis.

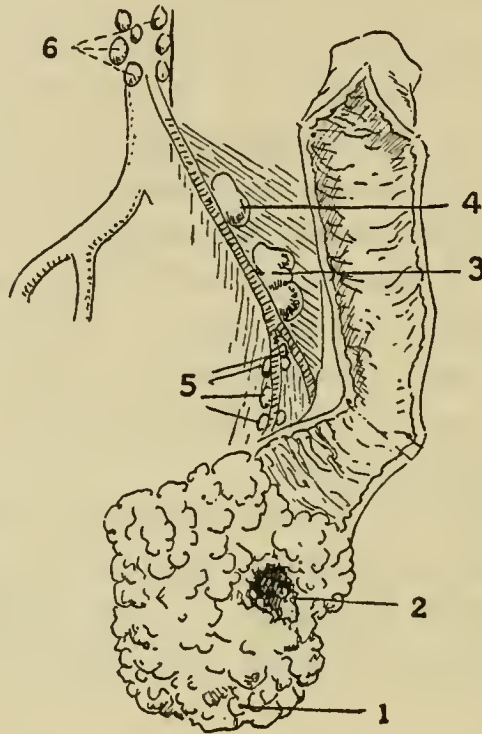
It is difficult to describe the precise characters pertaining to these three forms in a rigorously differential description, because the growth may be infiltrating or budding, confined at one surface of the rectum or be annular, pedunculated or implanted in a large base, ulcerated or not. The ulcer may be small or very extensive, with flat or very granulating edges, with a hollow or exuberant fundus and a narrow or broad base. Lastly, the stenosis is more or less tight, more or less long with infiltrated or ulcerated walls.

Nevertheless, I shall select the simpler cases for demonstration and will endeavor to show the distinctive signs, but always recalling that at the level of the mucosa the tumor is only a temporary phase of the process and that a cancer invariably ends by ulcerating.

*Tumor.*—A non-ulcerated malignant tumor of the rectum forms a more or less marked projection into the lumen of the bowel, hence

is more or less obstructing. Occasionally the neoplasm is implanted on a rounded and distinctly limited base, raising up the mucosa by small unequal mammillated buds closely packed together, or by a single tumor mass having a hemispherical outline. The color is a reddish violet or bright red, the consistency hard. The following is the description of such a growth (see Fig. 1).

FIG. 1.



1—Anus. 2—Ulcer. 3—Large multiple glandular mass at bifurcation of superior hemorrhoidal artery. 4—Large lymph-node on the inferior mesenteric artery. 5—Lymph-nodes lying on the superior hemorrhoidal vessels. 6—Small preaortic lymph-nodes.

The specimen came from a patient operated on by Hartmann by the abdomino-perineal method, on July 25, 1912.

Thirty centimetres of bowel were excised. The specimen was opened longitudinally on its right lateral aspect, presenting the neoplasm in front and the retro-rectal tissue immediately behind.

The tumor projects into the lumen of the rectum at a distance of fifteen centimetres above the anus. Its breadth is seven centimetres. It is budding, the buds being closely packed together. There is no ulceration, but the mucosa is wrinkled, violet in color and traversed by two or three fine sulci. The edges of the tumor are raised about one and a half centimetres, irregularly rounded and jagged. The growth is not exactly circumferential but rather in a left postero-

lateral position, extending slightly onto the anterior aspect of the rectum. Above and below the growth the mucosa is soft, pliable, not ulcerated, without polypi and of a good color.

The retro-rectal tissue is thick, composed of hard, dense, lumpy fat without any trace of neoplastic infiltration and for that matter, the external surface of the rectum is intact. The course of the vessels in this tissue could be dissected; they were large and rigid. The bifurcation of the superior hemorrhoidal corresponded to the lower edge of the tumor and at this level there were three enlarged lymph-nodes the size of a filbert, hard to the feel and white on section. Six to eight lymph-nodes of about the same size were grouped around the blood-vessels above, in pairs or single.

This lymph-node chain began above at about five centimetres below the ligature of the arteries and ceased exactly at the level of the arterial bifurcation. The upper and lower limits of the chain corresponded to those of the tumor. By following the branches of the superior hemorrhoidal artery I was unable to find a single lymph-node higher up.

*Histological Examination.*—Typical adenocarcinoma; no visible neoplastic emboli could be detected in the lymph-nodes; they were in a state of subacute inflammation.

Occasionally the mucosa will present small areas of ulceration when the growth is very budding and in these irregular fissures a fetid mucopurulent stagnates, as well as débris of slough, old blood and adherent mucus. After washing the tumor it will appear red or pink, with marked exuberant vegetations and riddled with fissures or presents a single small crater.

A more or less voluminous cancer, hence more or less heavy, may drag on its implantation and elongate its base until a kind of pedicle is formed, or it may produce prolapse of the corresponding rectal wall from the weight of soft or hard, irregular, bleeding buds. All degrees of this secondary prolapsus can be met with, from prolapse of the tumor to prolapse of all the rectal walls.

*Ulceration.*—In some cases the ulceration is very limited in extent and presents the classic anatomical characters of cancerous ulcerations—raised, everted, indurated and jagged edges; a crateriform or vegetating fundus, the latter red, bleeding or sloughing, the former

black, unpolished and fetid. The mucosa stops irregularly at the edge of the ulcer. The following is a good illustration (see Fig. 2).

This specimen was obtained from a male operated on by Hartmann by the abdomino-perineal route.

The segment of bowel removed measures twenty-three centimetres. Examination of the recto-anal canal incised along the anterior wall:

FIG. 2.



At the lower part the smooth, soft mucosa was raised up by small hemorrhoidal tumefactions. Three centimetres above the ano-cutaneous line was a round ulcer seated on the right side of the ampulla and reaching the middle line on the posterior aspect. It measures four centimetres transversally and perpendicularly; this circumference was slightly festooned, the raised edges very hard to the feel and everted especially at the upper and lower parts of the lesion. They were red on the side of the ulcer, livid on the side of the mucosa.

Between the edges and surrounding normal rectal mucosa was a sulcus about half a centimetre deep. The fundus of the ulcer was uneven, oozy, greenish, hard to the feel and not perforated. On the left of the ulcer was a budding mass, slightly pedunculated, the size of an almond with a surface irregularly covered by fissures;

below it was livid white with bluish streaks while above it was wine-colored. Above, the mucosa of the ampulla was normal in color, but two centimetres above the upper edge of the ulcer the posterior rectal wall was raised up by an oval tumefaction the size of an almond which flattened out the mucosa so that it could not be picked up at this point.

When the incised bowel was closed it was found that the bud formation became embedded in the ulcer, that there was no macroscopical lesion at the point of reflexion of the peritoneum and that above the ulcer the rectal walls were very thin, almost transparent.

Examination of the *mesorectum* showed that behind the rectum there was a thick layer of fatty tissue, brown in color and soft in consistency below, while above it was yellow and harder. *Towards the middle part of the posterior aspect of the ampulla and forming one body with it, was a budding mass very hard to the feel and solidly implanted.* It was this production that projected under the intestinal mucosa. Four centimetres above there was a mass of confluent lymph-nodes the size of a large walnut situated on the course of the superior hemorrhoidal artery, six centimetres below the point at which the vessel had been ligated. It was impossible to dissect out the vessel from this mass, which bifurcated at this point. The lymph-nodes situated the highest up was fourteen centimetres above the upper edge of the neoplasm.

*Histologically* the growth was an adenocarcinoma; the adherent retro-rectal mass was a cancerous gland with its shell ruptured. There were no visible cancerous emboli in the high situated lymph-nodes, only a subinflammatory state.

In other cases the ulceration is extensive and its limits ill-defined. The fundus is covered with large vegetating red buds, very vivacious and which, by falling over on the side, may hide the limits of the ulcer. This is the so-called "ulcer-tumor" so characteristic of intestinal cancer.

The shape of the lesion is still more variable. The ulcer may extend in surface or in depth; the edges may be flat or hypertrophic; there may be fissures. The process of ulceration in rectal cancers almost invariably ensues.

*The Annular Form.*—There is a form of rectal cancer that from

the very early phase becomes circumferential producing a straight central annular stricture. The rectal walls are thickened by a tumor which does not project very much but is very infiltrating. The intestinal lumen is sometimes insufficient to allow the little finger to pass through, the stricture is more or less tight with hard inextensible walls. After longitudinal division of the rectum and spreading out the specimen there will be seen either small buds packed together and projecting under an almost normal mucosa or a circular ulcer not very elevated or simply a ring-shaped, sharp or rigid cylinder-like mass projecting under a folded wrinkled mucosa. Unquestionably this is the so-called scirrhus form of rectal carcinoma, that is to say, an infiltrating cancer against which the organism sets up a defensive action of the tissues by intense sclerosis.

The stenosis is perhaps more the result of the rectal sclerosis infiltrated with cancerous cells than to the neoplasm itself. It is a kind of *plastic proctitis* of the rectum. The following is given as an illustration of a typical annular cancer (see Fig. 3).

The specimen came from a male operated on by the abdomino-perineal route by Lardennois.

The segment removed measured twenty-five centimetres in length. Looking at its external aspect the intestine appeared to be contracted towards its middle part at about thirteen centimetres above the anus. A ring-shaped sulcus appeared to exist at which point the uninvaded serosa seemed to be shrunken. Above, the intestine was distended and by palpation its walls were thick, solid and hard.

Below the stenosis the walls were lax and not very thick. With the water-test the pocket above the stricture became distended and the water passed very slowly through the lower segment. A grooved director could just pass through the stenosis.

The intestine was opened longitudinally on its anterior aspect and a very tight stricture was found fourteen centimetres above the anus. At its level there was a rigid, inextensible, slightly ulcerated and exactly circumferential tumor, about one and a half centimetres in height, reposing on a very hard base. The normal mucosa abruptly ceased at the edges of a narrow ulcer having the shape of a stellate fissure. Its edges, which were not much raised, were dark red. In the deep cut ulcer there was a shred of slough. Above and below, the

mucosa was pink and soft. There were two small polypi, one three centimetres, the other six centimetres above the neoplasm. Immediately above the stenosis was a dilated pouch whose fundus dropped down on the right below the level of the stenosis. This almost diverticular dilatation could contain a tangerine orange; its mucosa was blackish and, as in the entire upper segment of the bowel, the walls were extremely thick—one to one and a half centimetres—and very hard. Below the stricture, the intestinal tunics were soft and flexible. At the level of the stenosing growth, the circumference of the rectum, when the specimen was spread out, measured about four centimetres, while that of the pouch above was about twenty-five centimetres.

The mesorectum was filled with much soft fat, the dissection of the vessels was difficult and the bifurcation of the superior hemorrhoidal artery took place one centimetre above the rectal stenosis. At this level there were two lymph-nodes the size of peas, while above, along the course of the vessels there was a chain of four or five hard small lymph-nodes, one of which was situated two centimetres above the bifurcation of the artery. It was the size of a hazelnut and macroscopically was neoplastic.

Histologically the neoplasm was an adenocarcinoma freely infiltrating the rectal walls. In the lymph-node incised there was a very distinct tuberculous follicle but no trace of cancerous invasion.

Such are the aspects of cancers of the rectum, but each neoplasm has its own physiognomy and the typical cases of tumors, ulcerations, and stenosis are not very frequent. Most usually it is an infiltrating budding tumor, ulcerated, bleeding, obstructing, and by its size or circular extension develops into a long, anfractuous and tortuous stricture. It may at times be difficult to recognize on a vegetating growth whether an ulceration has secondarily developed or whether voluminous buds have developed at the bottom of a long-standing ulcer.

It is only by referring to the most simple cases, all histologically verified, like the examples I have given, that I have thought that the oldest yet simplest classification could be retained. The proctoscope and early operations will certainly reveal the initial phases of cancer of the rectum.

Some observers have thought that ano-rectal cancers often assume the aspect of a projecting tumor, that ampullar cancers are often ulcers and lastly, that the annular form is a high-situated neoplasm, that is to say, above the ampulla.

For example, Funke has observed sixty-four cases of high-seated cancer and Zinner thirty-four. Nevertheless these growths were annular and in twenty-seven out of the thirty-four cases of Zinner there was a stenosis.

To sum up, only now considering the morphology of rectal cancer from *the viewpoint of macroscopical pathology alone*, I believe that at a very early phase in its evolution, cancer of the rectum almost always circumscribed, may appear as a tumor, as an ulcer or as a more or less stenosing ring. There are other anatomical forms, almost all resulting from complications, such as prolapsed cancer, suppurating or fistulous cancer, etc.

*Extent.*—Tumors that have become very voluminous either form an enormous ascending or descending or prolapsed projection into the rectum, or else massive cancers, cause an extensive infiltration of the walls of the rectum transforming the organ into a thick, rigid cylinder as hard as wood.

The ulcerations may develop in height—from two to fifteen centimetres—but usually, according to the law of cancer extension along the lymphatic channels, the lesions of the rectum tend to become circular. It is true that those which to digital examination appear to be very exactly circumferential, will show a healthy area on the opposite side of the rectum to that of neoplasm when the specimen has been opened and spread out. Sometimes in order to recognize this intact area the broad everted edges of an invading ulcer must be raised up.

The tendency of cancer of the rectum to become circular may be estimated by the following statistics: Out of a total of 116 ampullar cancers Zinner found 59 circular growths, or 50.8 per cent. Out of a total of 80 ampullar cancers Lorenz found 34 circular tumors, or 42 per cent.

According to Quénu and Hartmann one frequently meets with secondary nuclei, either opposite the growth, or above or below it. The former by uniting with the primary growth favors an annular

production. I have given the description of a secondary focus opposite a primary cancerous ulcer. The following is the case of a patient seen by Hartmann in whom clinical examination revealed a secondary focus, verified by macroscopical and microscopical examination.

Female, *æ*t. 59 years, has been constipated for a long time. For the past three or four months red or black blood has been present in the stools, nocturnal sacral pain. For five or six days the stools have been frequent with alternating diarrhœa and constipation.

By digital rectal examination a tumor was discovered commencing in the anal canal, occupying the left lateral half of the ampulla and ceasing at the anterior median line but extended beyond the posterior median line. Its edges were raised, the mucosa surrounding it soft and pliant. Nevertheless on the right in the healthy area one could feel a nodule embedded in the mucosa. The examination caused the discharge of a bloody, serous, fetid fluid. No enlarged inguinal lymph-nodes.

*Biopsy.*—The isolated nodule on the right side proved to be microscopically a cylindrical epithelioma.

Perineal excision of the rectum was done by Hartmann. During the operation large lymph-nodes could be felt in the mesorectum in front of the sacrum. They were excised.

Cancer of the rectum is very prone to extend into the perirectal cellular atmosphere, rather than into the walls of the bowel.

*The State of the Bowel above the Neoplasm.*—It is above all when an annular cancer causes a more or less marked stenosis of the lumen of the rectum that important changes ensue in the intestine above the growth. Or on the other hand, the muscular layer is thickened and vigorous, indicating its long efforts at contraction and becoming hypertrophied from the extra work put upon it. In these circumstances the thickness of the walls above the stenosis may reach one centimetre and extends upwards as much as twenty centimetres.

But thickening of the walls may be simply due to œdema, either mechanical or inflammatory, and thus is explained the thick, lax and friable walls encountered. Or the tunics of the gut above the stenosis may give way with the result that a dilated pocket results, even when the hypertrophied muscular layer might be supposed more

resistant. Usually, however, this dilatation is the result of an insufficiency of the walls which are thin. Such a dilatation was very well seen by radiography in one of my cases.

But still more serious lesions may exist in the bowel above the rectal cancer, namely, ulceration and bifurcation, which I shall now consider.

*Ulceration.*—Curling says that he had seen a patient and a specimen taken from another in which the physician, while using bougies for dilating the stenosis, had entered the abdominal cavity through the softened rectal structures and peritonitis ensued. Mollière refers to a specimen removed at autopsy in which the thinness of the walls above the stricture was such that a very slight pressure with the finger was enough to produce a tear in the intestine opening into the peritoneal cavity.

Chalier and Thomasset have given a description of ulcers distant from the cancer. They collected seventeen instances of lesions of the bowel above the rectal cancer, but they believe that these are more frequent than what the small number of published cases would lead one to assume. Multiple ulcers of the intestinal mucosa of dimensions varying from the size of a pin's head to a silver dollar may exist; they are oval or round with irregular, polycyclical or sharp edges and sometimes present a honey-combed aspect. They are more prone to be seated on the left side of the large intestine, but in one of Kocher's cases they were disseminated in the small intestine.

Letulle terms them dysenteriform ulcerations and states that there is a veritable symptomatic dysentery, partial and localized above an incomplete and chronic obstruction of the large intestine.

The cause of these ulcerations is a moot subject. For some, they are the result of the mechanical action of the hardened feces which stagnate above the stenosis. But according to Kocher, these ulcerations may be seated at points where the feces are soft. Like Cavaillon and Leclerc, Chalier and Thomasset invoke microbic pullulation and readily adopt the infectious origin of these ulcers. According to Kocher, the distension of the intestine by fluids and gas brings about a venous stasis which first of all manifests itself in the corresponding mesenteric segment, provoking ecchymoses, followed by shedding of the poorly nourished epithelium and finally a superficial necrosis of

the mucosa ensues. In fact he points out that in strangulated hernia the ulcers are always seated on the convex border of the intestine where distension is the greatest.

Voscaï Shimodaira recently reported forty-eight cases of intestinal stenosis with ulcerations especially seated in the large intestine, as well as the results of experimental work on twenty-four dogs. He believes that intestinal ulcerations may be the result of distension alone. A severe distension stops the arterial circulation and brings about anæmic gangrene of the walls. A moderate distension is accompanied by vascular paralysis and a severe venous, hyperæmia which may provoke gangrene. Ulcerations and perforation occur at the point of necrosis. It has seemed to this observer that the bacteria and toxins of the intestinal contents had no influence whatsoever in the production of ulceration, but he, however, admits that if distension of the gut creates ulceration then the bacteria may cause it to extend.

In the specimens of rectal cancer that I have studied I did not find any ulcerations, but Hartmann has seen them on the pelvic colon in a case in which he did a left iliac colostomy for rectal cancer, and Lecène during a laparotomy for intestinal occlusion discovered a recto-sigmoid cancer above which there was a considerable dilatation of the large intestine. The tranverse colon having been accidentally nicked, it was used for the colostomy.

*Perforations.*—Perforation of the intestine may occur near to or distant from the neoplasm. When it takes place close to the neoplasm, the perforation is more apt to result in abscess formation but when it is distant peritonitis ensues. Chalièr and Thomasset have collected thirteen instances of distant perforation with the following sites:

Cæcum .....	3 cases
Ascending colon .....	1 case
Transverse and descending colons .....	2 cases
Pelvic colons .....	6 cases
Small intestine .....	1 case

These perforations may be traumatic. Kocher's well-known case was that of a patient with a left colostomy who died of peritonitis due to peritonitis from perforation, the latter being caused by irrigations of the colon. A similar instance occurred in Hartmann's practice.

Female, *æt.* 61 years, had suffered since November 26, 1909, from colic. During this time she was seized by bilious vomiting and also noted blood in the stools. The pain was seated in the sacral region, was more severe when the patient was lying down or seated, and diminished when the patient walked or went up stairs.

Digital examination revealed an ampullar cancer through which the finger could not pass.

On March 9, 1909, an attempt was made to remove the rectum by the peritoneal route. During anterior peeling off of the rectum—the cancer was adherent to the vagina—perforation of the bowel occurred, while posterior detachment was found impossible. The operation was stopped, the incisions packed with gauze and a drain placed in the presacral region. Left iliac colostomy.

On April 15, a large lavage was done through the colostomy removing a large amount of feces, immediately followed by violent pain in the right iliac region. On April 17, the general condition of the patient was very bad with abdominal distension, rigidity over the right iliac fossa and pain over MacBurney's point. Pulse small and rapid. Patient died on April 19, with progressively increasing abdominal phenomena.

More frequently these perforations are spontaneous. Some occur from bursting of the gut—*diastatic perforation*; others result from the progress of the ulceration—*gangrenous perforation*.

Diastatic ruptures are the most interesting, cases having been recorded by Zauer, Kocher, Dufourmentel and Lyot. The latter observer distinctly notes that the mucosa was healthy and that the appearance of the perforation was that of a traumatic rupture.

But Chalier and Thomasset's case is still more instructive. The patient had an occlusion and in view to doing a colostomy the incision was made on the right. The greatly distended cæcum exteriorized itself and on its external longitudinal band at its junction with the ascending colon, a star-shaped diastasis of the serosa was perceived on the point of perforation. The peritoneal diastasis was completely buried by several sero-serous sutures and colostomy was done in the immediate neighborhood. The outcome of the operation—which certainly prevented peritonitis—was in every way satisfactory.

Auschutz carried out an experimental study of diastatic perforations on the cadaver and he came to the following conclusions: (1) The weakest point in the small intestine is at the level of the insertion of the mesentery; (2) in the colon the rupture always takes place at the union of the ascending colon with the cæcum near the anterior band; (3) rupture always takes place as follows: The peritoneum is the first to tear parallelly to the axis of the intestine, which is explained by its lesser elasticity, then the muscle gives way and lastly the mucosa. The tear in the muscle is usually transversal.

Perhaps the following case, taken from Hartmann's service, may be an example of perforation by diastasis.

Female, *æt.* 40 years, entered hospital for frequent attacks of intestinal obstruction which were no longer relieved by enemata and aperients. Digital examination of the rectum revealed an uneven mass very high up, voluminous, knotty, hard and fixed on the anterior wall of the rectum.

A few days after there was very great distention and no gas was passed, so that an incision on the right side was made with a view to enterotomy. When the peritoneum was incised fetid gas escaped and the abdomen progressively collapsed. The intestinal coils were agglutinated. An intestinal coil near the abdominal incision was perforated and gave exit to intestinal liquid. On the following day the pulse was very rapid and the facial expression that of peritonitis. The patient was discharged at her request.

Gangrenous perforations especially occur on the left side of the colon; their edges are ragged and gangrenous. A perforation of one of the ulcers is common and is the final phase of the process of ulceration seated above the rectal stenosis.

*The Adenopathies.*—The studies carried out by Kuttner and Poirier on the lymphatic invasion in lingual cancer, and those of Cuneo on lymphatic invasion in gastric and laryngeal cancer have been the means of great progress in the surgery of the lesions of these organs. If, as Lecéne has justly said, malignant tumors are to be operated on as soon as possible, far beyond the apparent limits of the malignant process and if possible removing the first lymphatics involved, it is essential to precisely know as far as possible the limits

of the initial growth and the site and usual limits of its immediate adenopathies.

I have dissected thirty specimens with the view of determining which lymph-nodes cancer of the rectum invades at the onset and above all which are the ones that should be removed in order that the operation shall obey the general principles of the surgery of cancer.

The specimens at my disposal were derived from seventeen autopsies made with the special purpose of studying the lymph-nodes and twenty-three very extensive abdomino-perineal excisions of the rectum.

With the exception of three cases the ano-rectal and superior hemorrhoidal lymph-nodes were enlarged. It is not indifferent to add that in one of these cases there was an interposed lymph-node which was involved neoplastically—a para-vesical lymph-node, proved by microscopical examination.

In seven autopsies I have searched for the middle hemorrhoidal lymph-nodes without success and in only one case were the internal iliac lymph-nodes increased in size. Finally, in numerous case histories I have found mention of enlargements of the inguinal lymph-nodes, but it seems to me proved that this can exist in cylindrical epitheliomata even when situated very high up and that consequently their presence could not especially indicate the pavement-cell nature of the growth or even the invasion of the anus by a cylindrical-cell neoplasm.

I would say at once that an increase in the size of the lymph-nodes does not appear to me to belong to certain macroscopical forms of growth more than to others, and I would insist upon the fact that around non-ulcerated tumors I have met with large lymph-nodes, while in very deep ulcers, even perforating and apparently infected I have only found very small nodules. I mention this to show that those cancers the most exposed to infection are not of necessity complicated by large adenopathies. However, cancers situated high up seem to become rapidly complicated by large retro-rectal adenopathies.

*The Inguinal Lymph-nodes.*—These are enlarged in cancer of the anus. The adenopathy may be unilateral if the ulceration is likewise frankly unilateral.

The adenopathy may be overlooked even when a methodical examination of the patient is made. This homolateral unilaterality and latency of the inguinal invasion are well proven by the case of a female, *æt.* 48 years, upon whom Lecène excised an ulceration on the right side of the anus. One year later a recurrence took place in the right inguinal lymph-nodes. These were excised and the patient lived for a long time afterwards.

More often the adenopathy is bilateral. In a case recently seen in Morestin's service, the lymph-nodes on the left side were few but very large. On the right, they were small, very numerous, painless and invaded the skin. They were connected with the malignant growth by a very dense perineo-crural lymphangitis. In a patient seen in Walther's service, there was in each inguinal region large masses of lymph-nodes clinically inflammatory—painful, fluctuating and finally giving rise to fistulæ.

This inguinal adenopathy may exist in cases of cancer situated high up in the rectum although neither the anus nor sphincter are involved in the process. It is mentioned by Kocher in three cases of tumors seated from four to six centimetres above the anus and in two cases reported by Chalièr, one an ampullar cancer, the second a cancer above the ampulla; there were large lymph-nodes in both groins. Villard's statistics contain one of a cancer seated eight centimetres above the anus with a left-sided inguinal adenopathy and two instances of bilateral inguinal adenopathy in a case of ampullar cancer and one in which the growth was seated above the ampulla.

I have found several cases of inguinal adenopathy in high-seated rectal cancer the precise nature of the lymphatic enlargement was not ascertained microscopically. The diagnostic value of these external adenopathies in rectal cancer is still a moot subject. Nevertheless I believe that enlarged inguinal lymph-nodes in cancer of the rectum may occur much more frequently than is generally supposed in cases where the neoplasm is seated high up. In point of fact there are rich lymphatic anastomoses between the lymphatics of the anus and rectum.

(2) *The Middle Hemorrhoidal Lymph-nodes.*—Quénu discovered the middle hemorrhoidal lymph-node at the point where the internal iliac artery gives off its cluster of branches or on the lateral wall of

the pelvis at the level of the sciatic notch. According to Cunéo and Marcille, the middle hemorrhoidal lymph-node is situated some distance from the lateral wall of the pelvis on the course of the artery, while Boulay states that on the course of the middle hemorrhoidal artery small lymph-nodes, varying in number, exist—Cunéo and Marcilles lymph-nodes; the principal trunks continue towards a larger lymph-node situated at the emergence of the middle hemorrhoidal artery—Quénu's lymph-node.

Cancerous metastases in these lymph-nodes appear to be exceptional or else very difficult to find. It is true that Fayard has mentioned a cluster of lymph-nodes situated under the peritoneum in a case of rectal cancer; they were on the outer side of the rectum and inner side of the branches of the hypogastric artery and the pelvic area corresponding to the cotyloid cavity. He calls it the pelvic group, believes it to be frequently involved in cancer and points out the difficulties to explore it. Carl Koch has also mentioned this group of glands and Quénu and Hartmann have verified their malignant invasion both in the living and at autopsy.

I have searched for these adenopathies by dissection in seven autopsies on subjects presenting very large malignant neoplasms of the rectum and have failed to find them. On the other hand, I have been unable to find in medical literature—providing care be taken to carefully distinguish these lymph-nodes from the internal iliac lymph-nodes—a single case which clearly proves their invasion by cancer or a single drawing exactly giving their topography and indicating their importance.

(3) *The Ano-rectal and Superior Hemorrhoidal Lymph-nodes.* The constancy of a solitary or predominating superior hemorrhoidal adenopathy, symptomatic of high or low rectal cancer allows one to conclude that it is the immediate most important and most surgical adenopathy of cancer of the rectum (see Fig. 4).

Enlarged lymph-nodes always exist in the retro-rectal tissue, sometimes easy to dissect out of the soft fat, at other times embedded in inflammatory sclerous tissue or strangled by cancerous periproctitis. They are to be found along the lateral superior hemorrhoidal arteries in more or less compact chains, to the outer or inner side of the vessels, being more retro-rectal than latero-rectal.

The lowest of them are always opposite the tumor; *exceptionally* *them may be lower than the site of the neoplasm* and quite often they are above it. In a case of cancer developing in an anal polypus there were three enlarged lymph-nodes just above the sphincter, while in another case—an ano-rectal ulcer—the first lymph-node, which was very large, was found ten centimetres above the anus.

They vary in size from a pea to an almond; they are round or oval, but if their shell has ruptured they are irregular. I have never found them softened or suppurating; their consistency is more or less firm, while on section they offer a pink or yellow white surface, sometimes gray or blackish.

*But I would insist upon the following fact, namely, that there always exists a group of lymph-nodes—two, three, four or more—exactly at the level of the bifurcation of the superior hemorrhoidal artery.* If there are enlarged lymph-nodes along its terminal branches—Gerota's ano-rectal lymph-nodes—the group at the bifurcation is always the most important of the two. Enlarged lymph-nodes may only exist here as in the case of a patient operated on by Hartmann by the abdomino-perineal route and in whom retro-rectal dissection only revealed four lymph-nodes the size of filberts closely clustered around the bifurcation of the artery. In other cases, such as the one depicted in Fig. 1, a high situated rectal cancer may give rise to an adenopathy high up along the terminal portion of the superior hemorrhoidal trunk and it is still at the level of the bifurcation that the largest lymph-node was found.

Finally, in some cases two chains of lymphatics extend along the lateral superior hemorrhoidal arteries, their lymph-nodes progressively increasing in size from below upwards until the principal lymph-node at the bifurcation is reached, as shown in Fig. 4.

Hence there always exist ano-rectal lymph-nodes lying against the visceral musculature in cancer of the rectum, but the principal group will invariably be found at the bifurcation of the superior hemorrhoidal artery.

At an autopsy on a case of very extensive ano-rectal cancer adherent to the prostate and bladder I found that the only adenopathy was in the retro-rectal territory and along the course of the inferior mesenteric artery (see Fig. 1).

*Description of the Autopsy Findings.*—The specimen was taken from a male who died in the service of Doctor Riche, at the Tenon Hospital.

The colon was empty, the iliac colostomy was functionally good. From the splenic flexure up to the colostomy, the colon was small, hard and with thick walls. The ascending colon was small and hard with a dilatation below the liver and a still greater dilatation of the cæcum. The cæcum was very distended and its walls transparent. At no point were ulcerations present in the intestinal mucosa. Preaortic lymph-nodes were found below the renal pedicles (see Fig. 6). The specimen was removed *en masse* by pelvic evisceration, including the bladder, rectum, iliac vessels and perivisceral sheaths. The inferior mesenteric artery in its entire length and the lower part of the abdominal aorta were attached to the specimen.

On the right and left of the aorta, from the renal arteries to the origin of the inferior mesenteric artery were five lymph-nodes the size of a hazelnut on each side; they were not very hard and were dark red in color.

On the course of the inferior mesenteric artery, midway between its origin and bifurcation into the two superior hemorrhoidals, was a large lymph-node, very hard, regularly rounded and whitish in color.

In the angle formed by the bifurcation of the superior hemorrhoidal were three or four small lymph-nodes the size of a pea, but situated at one centimetre above them was a large polyglandular mass, irregular in shape, budding and adherent to the artery (see Fig. 1, 3).

There was a small chain of lymph-nodes close to the right superior hemorrhoidal artery. There were no lymph-nodes visible along the iliac vessels or middle hemorrhoidal arteries.

The rectum in its ampullar segment formed a mass the size of an orange. The growth could not be seen from the anus. The bowel was opened along its left lateral aspect revealing a large, budding tumor, occupying the anal canal and the two lower thirds of the ampulla. The neoplasm was about ten centimetres in height, perfectly circular throughout its extent, with soft ragged edges below and indurated, everted bright red edges above. A small deep crateriform ulceration was seated at the ano-rectal junction. In front, no

FIG. 3.



Cancer of rectum.

FIG. 4.



Cancer of rectum.

FIG. 5.



Cancer of rectum.

FIG. 6.



Cancer of rectum.

FIG. 7.



Cancer of rectum.



line of cleavage could be found for separating the rectum from the bladder or prostate.

When the bladder was opened a large retention of urine was found and in the area of the trigonum the wall of the bladder was pushed in and its mucosa was in a state of congestion. The ureteral openings were patent.

Verification of the pelvic cavity showed that all the structures had been removed with the vesico-rectal mass. No lymph-nodes were found in front of the sacrum or at the superior strait of the pelvis.

Anatomists do not insist upon the importance of the group of lymph-nodes at the bifurcation of the superior hemorrhoidal artery. Another very important fact is that the lymphatics of the cutaneous anal area attain the lymph-nodes existing at about the level of the second sacral vertebra.

Trélat and Delens maintained that enlargement of the retro-rectal lymph-nodes rarely occurred. This was true at a time when extensive operations were not undertaken, but at present this no longer obtains. *Histologically these high-situated lymph-nodes, ten to fifteen centimetres distant from the neoplasm, may be decidedly neoplastic so that another malignant neoplasm may develop in them and in turn involve the rectum more or less high up.*

*Briefly, the invasion of the lymph-nodes in cancer of the rectum particularly attacks the ano-rectal nodes and above all, an important group which has not before been described situated at the level of the bifurcation of the superior hemorrhoidal artery where the veritable arterial and lymphatic hilum of the rectum appears to be located.*

Involvement of the middle hemorrhoidal lymph-nodes has only been exceptionally encountered. That of the inguinal lymph-nodes is not uncommon. It usually takes place in cancer of the anus and may also occur in cylindrical-cell cancer situated high up.

Finally, in some cases, all these lymph-nodes may be invaded, but their macroscopical characters will never allow one to decide whether they are the site of an inflammatory or a neoplastic process.

Among the many specimens it has been my lot to examine, I will briefly report two as they well illustrate certain conditions met with.

The first one (see Fig. 2) is that of an excision of the rectum thirty centimetres in length performed by Cunéo. At its lower

part the rectum is greatly increased in size and appears to be filled with a hard mass. The mesorectum is not very thick. The bowel was opened on its anterior aspect. At the lower part of the ampulla is a tumor the size of an apple implanted on a relatively small, indurated base about the diameter of a one dollar silver piece. The tumor was very projecting with irregular prolongations, some ascending, others descending.

The neoplasm is soft, its surface covered with elongated papillæ which are united together at their apices. The rectal mucosa is normal above the growth. The mesorectum contains fluid fat. By dissection four to five small lymph-nodes were found immediately above the neoplasm and three lymph-nodes the size of filberts were discovered at the bifurcation of the superior hemorrhoidal artery. When the specimen is placed in formol the prolongations become detached from each other and float in the solution like villousities.

*Histological Examination.*—Papilliferous adenocarcinoma. The enlarged lymph-nodes offered a vulgar hyperplasia and no malignant change.

The second specimen is a low-seated cancer of the rectum and recto-colonic polyposis. The specimen, from a patient operated on by Lardennois (see Fig. 5), measured forty-five centimetres in length. The bowel was divided at operation just above the sphincter. The intestine was opened on its anterior aspect. Its cavity was found riddled with polypi throughout its entire length, and the point of division of the bowel above was made in the polypous area. These small pedunculated tumors were raspberry red, were very near together, almost confluent at the lower part of the gut, and some of them were the size of an almond. Between the polypi the mucosa was normal and had preserved its folds.

At the very bottom of the specimen, divided by the longitudinal section of the gut, was a small, hard whitish-yellow tumor, brilliant on section, infiltrating the walls of the intestine by a broad base. Its surface was vegetating but was not ulcerated. It was situated on the anterior surface of the lower part of the ampulla and in size was that of a silver quarter. The exposure and dissection of the vessels in the mesorectum were easy, and all along their course as far as the upper ligature small lymph-nodes could be seen.

*Histological Examination.*—Adenocarcinoma, not very infiltrating. It was an adenoma at the onset of malignant transformation. A lymph-node removed from behind the neoplasm was stuffed with cylindrical epitheliomata, hence revealing its malignant change. A lymph-node situated higher up was found to be simply hyperplastic.

*Distant Adenopathies.*—Cunéo has shown how the first lymphatic lymph-node clusters are the most important to know about. In fact it appears that cancerous emboli undergo a long arrest in the first lymph-node, but unfortunately this arrest of the epithelial cells is not definitive. The cancerous elements proliferate in the node, progressively changing its structure, and this protective organ fatally becomes, after a variable length of time, a new focus of infection, from which the neoplastic cells start off. As long as the first barrier of lymph-nodes has not been broken through, a radical surgical cure is still possible, but when this barrier has been forced the dissemination of the cancer elements into the lymphatic system makes a radical operation impossible.

And Cunéo goes on to make the following distinction:

“Under the name of immediate adenopathies I mean the degeneration of the lymph-nodes which are the first encountered by the lymphatics coming from the intestine. The name of distant adenopathies I reserve for infection of the lymph-node groups whose invasion follows the more or less complete degeneration of the first lymphatic barrier.”

Distant adenopathies have merely an anatomical interest; nevertheless an attempt should be made to clinically recognize them in order to avoid a useless surgical interference.

I believe that it is in the group of distant adenopathies that iliac adenopathies should be comprised, whether they are especially apparent at the level of the promontory or at the level of the lateral sacral notches. However, the lateral sacral lymph-nodes, that Bräitsef designates by the term of the central group of rectal lymphatics, may receive directly the middle hemorrhoidal collectors—Cunéo and Marcille—or the ano-rectal lymphatic trunks, although they represent the second barrier.

Theoretically, in fact, the cancer cells might invade—after the first barriers—the internal chain of external iliac lymph-nodes, the

lateral sacral group of the hypogastric lymph-nodes and, finally, the internal chain of the primary iliac nodes seated in front of or behind the vessels.

It has been said that a preliminary ligature of the hypogastric arteries, as the first step in abdomino-perineal excision of the rectum, would permit the excision of these iliac lymph-nodes. It has appeared to me that—unless it was an adenopathy along the middle hemorrhoidal artery—iliac metastasis strictly speaking is a sign of cancerous diffusion such that it should preclude the possibility of any radical operation. If it be the lateral sacral lymph-nodes that operative examination reveals, their excision can be easily accomplished during the process of retro-rectal detachment.

Lumbar adenopathies, either pre-aortic or on the sides of the aorta, have often been mentioned, and this condition I have seen. Finally, there are still more distant adenopathies. Halbron has published the case of a patient with a cylindrical epithelioma of the rectum and at autopsy a cylindrical epithelioma of the same type was found adherent to the stomach and developed in a lymph-node belonging to the gastro-omental chain. Nothing had caused one to suspect this growth secondary to cancer of the rectum.

Chalier relates the case of a female with a cancer of the posterior wall of the ampulla with lateral inguinal lymph-nodes, a lymph-node mass in the left iliac fossa and enlarged lymph-nodes in the left axilla.

Hartmann has published a case of cancer of the rectum whose histological structure was exactly reproduced in a supra-clavicular lymph-node on the left side.

Lastly, I would refer to Rabe's very interesting case. It was an autopsy of a man. In the pelvic cavity and at the bottom of the inferior peritoneal cul-de-sac emerged the peritoneal portion of the rectum visibly pathologically changed over a surface of about three centimetres and especially on the anterior aspect of its circumference.

The transparency of the peritoneum allowed one to follow the lines of lymphangitis which started from the diseased rectal wall behind and extended toward the sacrum and laterally along the branches of the hypogastric artery. Along the course of these lymphatics at intervals or distant from them, numerous lymph-nodes projected under the peritoneum. The mesorectum was infiltrated

by seven or eight lymph-nodes the size of peas. The lateral pelvic and sacral lymph-nodes were enlarged, and still higher up the cancerous adenopathy extended along the primary iliac arteries and on the sides of the abdominal aorta to the renal arteries.

Finally, from the angle formed by the internal and external iliac arteries, both on the left and right, the lymphatic vessels were seen to start following a retrograde course, accompanying the iliac vessels and becoming engaged with them under Poupart's ligament and then spreading out under the skin of Scarpa's triangle. These recurrent lesions were more marked on the left side. Hence is explained the presence of bilateral inguinal adenopathies and the existence of the resistant plane perceived on the left side and above the crural arch by deep palpation.

In this case one was dealing with a massive cancer of the rectum adherent to the bladder and prostate, with secondary deposits in the ureter and metastases in the liver. Consequently, it is understood that these were adenopathies of a pelvic cancer and not of a rectal cancer. On the other hand, nothing need lead one to suppose that the inguinal adenopathies were the result of a retrograde infection. But it is very interesting to note that the cancerous lymphangitis was seen going directly from the neoplastic focus to the lateral sacral and hypogastric lymph-nodes.

## COMPLETE PROLAPSE OF THE RECTUM

BY CHARLES J. DRUECK, M.D.

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COMPLETE prolapse of the rectum consists in the descent of all of the coats and is far more serious a condition than the partial variety because of the invalidism which it induces as well as the complications which are ever present. In this variety the mucous membrane is in its normal relation to the other coats of the bowel but the entire rectum is protruded from the anus and has lost its normal relationship to the other pelvic viscera (Fig. 1).

Two different types of pathologic change contribute to procidentia:

1. Extreme mobility of the rectum and the elongation of its supports may be the result of imperfect prenatal fixation or of traumatic conditions, either of which permit of constant dragging on the rectal attachments and supports. The intra-abdominal pressure exerted at stool is applied to the recto-sacral ligaments.

The uterus and rectum have a common means of suspension; therefore any cause bringing about the fall of one endangers the fixidity of the other. Hysterectomy deprives the rectum of the anterior support afforded it normally by the uterus. The weakening of the pelvic floor favors the prolapse of both of these organs.

The pelvic cavity is funnel shaped and from its lower opening protrudes the rectum held in place by the peri-rectal areolar tissue and fascia, the levator ani, the recto-coccygei, and the two sphincter muscles which are interleaved or woven into the pelvic fascia.

The pelvic fascia is a continuation of the lumbar, iliac and transversalis fasciæ and supports the abdominal contents from below. It is attached to the bony framework of the pelvis; in front to the inner surface of the pubic bone; on the sides, to the ilio-pectineal line, posteriorly, just above the attachment of the pyriformis, and to the anterior surface of the sacrum; and thus it binds the pelvic organs firmly together. From this level the fascia dips down between the pelvic organs forming the obturator fascia and the recto-vesical fascia, covering the levator muscle and also forming the deep layer

FIG. 1.



Complete prolapse of rectum.



of the triangular ligament. These structures form the true pelvic floor, but from these are projected extensions between and about all the pelvic organs which become accessory ligaments of these organs. The true pelvic floor is a fixed structure, but the fascial branches between these organs are suspensory stays allowing considerable play. It is these rectal stays which offer the resistance during the straining at stool which is necessary to prevent displacement of the rectum. When these stays become flabby from repeated or excessive stretching, they lose their contractile power and the organ they support drops away. A lacerated perineum destroys the fascia holding the rectum to the levator ani, and the powerful intra-rectal pressure soon pushes the rectal wall into the vaginal outlet. The protrusion in turn tends to further relax the musculo-fibrous structures.

2. In other cases a defect in the pelvic fascia permits a hernia of the pelvic bowel. This defect may sometimes be developmental.

In early embryological life the peritoneal pouch reaches almost to the perineum. Later it recedes higher and if this process stops early the cul-de-sac of Douglas will be deeper than is normal. Thus we may have congenital malformation of the sac as one of the factors in the origin of the hernia. If there is also a developmental defect in the transversalis fascia it requires but little increased intra-abdominal pressure to drive the peritoneum as a wedge along the prolongation of the transversalis fascia. This is the incipient stage of prolapse.

The peritoneal covering of the anterior wall of the rectum is very adherent to the deeper coats. The levator ani muscle and the very dense fascia on its lower surface also constitute a firm support to the perineal body and prevent a downward progression of the hernia. The line of least resistance seems to be through the muscular wall of the rectum, thus permitting the hernial development. The hernia now drives backward until it meets the resistance of the sacrum and coccyx when it is deflected downward through the rectal lumen, ultimately forcing the sphincters and appearing externally.

In every case presented, the condition of the sigmoid, the levator muscle and the depth of the cul-de-sac must be considered. An abnormally deep cul-de-sac acts as a pocket for the intestines which by their pneumatic pressure pry apart the musculature. In this

manner whenever the protrusion is two inches or more in length we may anticipate a fold of peritoneum, a coil of small intestine, an ovary or a part of the bladder wall to be included.

Several factors may contribute to the development of the prolapse and in the case at hand a combination of these may be found. Complete prolapse usually comes on slowly through long continued action of the primary cause, but in either children or adults it may come on suddenly as a result of severe straining during heavy lifting or as a result of a crushing accident or fall.

It may arise from tumor or stricture high in the rectum which causes persistent peristalsis or straining at stool. Ordinarily about three to six inches may appear although the whole colon and even part of the small intestine has been reported to protrude. Tillman cites a prolapse as large as a child's head.

When protrusion has taken place suddenly it may be constricted by the sphincter muscle and its reduction be difficult.

Three types or degrees of complete prolapse are usually described. The first degree closely resembles the incomplete prolapse beginning at the anal margin. Its external surface is continuous with the skin surrounding this aperture and the prolapse involves the anal canal together with a variable portion of the rectum.

In the second degree the prolapse begins at a point above the anus and the rectum is invaginated through the anal canal, which latter structure remains in position while the rectum protrudes externally. The walls of the anal canal are not here involved.

In the third degree some portion of the sigmoid or colon is invaginated into the rectum although it may not appear at the anus.

*Etiology.*—First Degree—This variety of prolapse results from the same class of causes as the procidentia mucosæ, and it is frequently a sequence of the latter. The distinguishing feature of this degree of prolapse is that the mucous folds which run up and down in the incomplete variety extend in a circular direction in the complete types and surround the prolapse in irregular crescentic folds.

The second and third degrees of prolapse represent the same character of pathology although the third type occurs higher in the bowel. Many factors may contribute to bring about prolapse such as elongation of the mesosigmoid, a relaxation of the sigmoid above the

level of the prolapse, an abnormally deep cul-de-sac into which the small intestines drop and by continued pneumatic pressure gradually work the levator ani and the pelvic floor away thus allowing the rectum to appear at the anus.

In this type the rectum invaginates through the anal canal and protrudes from the anal orifice, thus leaving a sulcus between the protruding rectal mucosa and the anal margin into which can be introduced a probe or sometimes the tip of the finger.

*Symptoms.*—The symptoms of complete prolapse are much the same as those of the incomplete type. The complete prolapse begins within the rectum and protrudes through the anal orifice, thus leaving a sulcus between the prolapsing gut and the anal margin. The differentiating feature of complete prolapse of small extent from an incomplete one of the same size is that the external surface of the protruding tumor is not continuous with the anal skin margin. There is a sulcus between the prolapse and the anal margin which is not found in the incomplete prolapse.

The protrusion is thick, firm and pyriform in shape, and when not more than three inches are present, the prolapse will extend straight out at right angle to the buttock with a slit-like orifice in the lower end. When more than this appears traction upon the mesorectum draws the tumor backward toward the coccyx and the orifice will be on the posterior surface. In exaggerated cases where the mesorectum and mesosigmoid are both dragged upon, the prolapse may make two or three corkscrew circuits. Sometimes in females the traction is forward because of vaginal attachments.

In old cases an hypertrophy of the exposed tissue occurs. All of the coats of the bowel are œdematous and swollen and often ulcerated. The mucous membrane is thick, dense and leathery in structure in the frequently prolapsed parts.

The surface of the mucous membrane is marked with circular furrows. The submucous areolar tissues are infiltrated with a hyaline substance, and the muscular layers are hypertrophied. The extruded part is therefore enlarged not only by œdema and congestion but also by the development of new structures. Therefore, the prolapse does not recede to its normal size when replaced, it is often too large to be retained, and descends the next time the bowels move.

In old or extreme cases replacement is difficult and painful, although gradually the anus becomes patulous and the sphincter so paralyzed that each time the sufferer defecates or even moves about, the mass protrudes and makes life a burden. The bowel is abnormally increased in size, and too large for its proper position within the pelvis, and although it may be reduced it will not remain so because the tenesmus set up by its presence expels it promptly. In some instances the mucous membrane is eroded and granular and easily bleeds. In such cases the odor of the sloughing tissues may simulate malignant disease. A proplapse that has protruded for some time is often accompanied with an oozing hemorrhage, which requires astringents to control. There is a copious discharge of glairy mucus which is often blood stained.

In children the procidentia occurs only at stool, but in aged persons with relaxed sphincters it may be down all the time. Constipation is the rule unless excoriation has occurred, when a teasing diarrhœa may be present. In either instance bloody and mucous discharges are present, and later fecal incontinence comes on. Pain is complained of only when there is ulceration of the prolapse or when spasm of the sphincters occurs which constricts the prolapsed bowel. Strangulation is present only in young and robust persons and is rare in infants or the aged. When it does occur it may be only temporary, but if it continues, ulceration and gangrene will follow which may terminate fatally if the peritoneum is involved. When the lower part of the rectum alone is involved in the gangrene, a spontaneous cure may take place, but by the separation of the protrusion and the resulting cicatrix a stricture is finally produced which leaves the patient in a more deplorable condition than before.

*Complications.*—Complications are prone to arise with the involvement of the peritoneal coat, for it is likely to carry down with it a loop of small intestine, an ovary or the bladder wall. When these organs are brought down, they are usually detected by touch and are generally found in the anterior part of the tumor. The intestine slips away from between the fingers with a gurgling sound due to the contained gas, or sometimes percussion demonstrates it by resonance. In the early stage the loops of the bowel are contained only in the anterior part. But if the protrusion is large the loops

may wholly surround the prolapsed bowel, except at the mesenteric attachment. In practice, if the buttocks are raised, the hernia usually recedes with a gurgling sound, and the prolapse may then be easily reduced. Adhesion between the loop of the small bowel and the prolapsed rectum may occur and strangulation result because the hernia cannot be reduced, or if the strangulation is not promptly relieved, death ensues from perforation of the bowel and peritonitis. If an ovary is included in the prolapse, pressure on it causes a faint sickening feeling, if the bladder is engaged it is demonstrated by introducing a sound through the urethra. Each condition constitutes a true hernia of the prolapse and must be immediately replaced, if possible, because spontaneous rupture of the rectal wall or of the peritoneal cul-de-sac and evisceration of the intestines has occurred and of course adds a most serious complication. Usually there is no sulcus or depressed line visible at the peritoneal or bladder junction with the bowel, and so there is no way of determining by inspection the presence or absence of peritoneum or bladder in the prolapse.

*Diagnosis.*—The differential diagnosis between the partial and the complete prolapse is often important. Prolapse of the mucous membrane alone is usually recent, the tumor is small sized, thin and soft to the touch, and the folds radiate from the orifice which is circular and patulous. When the deeper coats are involved the case is usually of long standing, the tumor is large and conical in shape, and its walls are thick and firm. The opening into the bowel is slit-like and usually points backward owing to the traction of the mesocolon, or points forward because of the vaginal attachments.

Hemorrhoids or neoplasms of the rectum which prolapse are differentiated by their irregular and lobulated shape and by finding other parts of the rectal circumference remaining *in situ*. Excoriation and hypertrophy resulting from the discharge may simulate epithelioma and may be differentiated only by a microscopical examination.

*Prolapse of the Upper Portion of the Rectum into the Lower (Invagination).*—By prolapse of the third degree is understood intussusception of the upper rectum, sigmoid or colon into the lower rectum or rectal ampulla. It is a true intussusception and may in-

volve any part of the large bowel even to the cæcum; the orifice of the appendix has been seen beside the included bowel. It differs from the ordinary type of intestinal intussusception in that it does not cause complete obstruction or strangulation. Also the approximating peritoneal coats do not adhere as they do in the intestinal intussusception.

In the previous types of prolapse the dislodged tissues protrude from the anus, but in this form the upper part slips into (telescopes) the lower part, the whole mass remaining within the pelvis. The sphincters and anal orifice remain normal. Only in extreme instances does the bowel protrude from the anus. When it does, it appears as a cylindrical tumor covered with a dark red, hyperæmic mucous membrane. There is no pain or soreness at the anus nor any sensation of protrusion at the anus.

*Symptoms.*—The symptoms of intussusception of the rectum or sigmoid are ill defined, because the rectum is capable of great distention in its lower portion. The invagination does not cause complete obstruction as in ordinary invagination of other portions of the bowel, nor do the peritoneal coats or the invaginated portion become adherent and fixed as they do in the upper portion of the bowel.

There is usually a history of protracted constipation and later an irregular diarrhœa accompanied with tenesmus, straining and a feeling of incomplete defecation. Laxatives are not effective, but much relief is obtained with enemas.

The liquid of the clyster lifts up the bowel from below and stimulates reverse peristalsis, thus disengaging the invaginated portion.

The immediate effect of this intussusception is obstruction of the bowels, but this is seldom complete because the feces are forced through by the increased contraction of the healthy bowel. The first symptom of the constriction is a sharp pain developing suddenly. It may pass off in a few hours to return again or it may continue from its onset. Vomiting sometimes occurs but not always, and if it does it is sometimes relieved by pressure. Abdominal tenderness may even be absent in some cases. The presence of fecal vomiting indicates complete obstruction regardless of the part of the bowel involved. A heavy dragging pain in the sacrum and radiating down the thighs

or to the perineum is usual. Dysuria also occurs and the case may be mistaken for ovarian or bladder disease. A discharge of clear mucus, later becoming tinged with blood, is present as the friction and irritation produces ulceration. If the constriction is severe enough, the prolapsed portion sloughs off and a circular cicatrix is left. Thus nature attempts to remedy the trouble, although the scar may produce an annular stricture. Sloughing frequently takes place after the first week and usually within three weeks, although it may occur much later. Death results in about one-half of the cases where spontaneous separation occurs, and may be due to one of several causes. The local peritonitis which unites the bowel may become general or the ensheathing portion, through ulceration and perforation may allow extravasation of feces. Perforation may occur at any weak point of union. On palpation, a tumor may be felt and may be characteristic although sometimes obscured by thick abdominal walls or distension of gases. The tumor when found is cylindrical and movable, even changing its position at times. Compared with obstruction of the upper bowels, intussusception of the colon or rectum is more chronic, less painful, diarrhœa is more pronounced, or the evacuations are larger, and the vomiting is variable. Such a condition may continue for weeks and death result from exhaustion or a general peritonitis.

Palpation or manipulation of the prolapse will often excite gurgling of the gas in the loops of small bowel which fill the anterior part of the prolapse. Percussion here will give a tympanitic note while the posterior half is dull on percussion. This condition is not found in the incomplete variety of prolapses, where only the mucous membrane is detached.

When the prolapse has been reduced a careful digital examination will note a laxity of all the rectal muscles and on palpation of the anterior rectal wall a distinct impulse will be observed on coughing as may be demonstrated in any hernia.

The introduced finger may feel the sulcus between the invaginated and invaginating parts. If beyond the reach of the fingers a probe may be used through a speculum. The examination for this purpose should be conducted with the patient upon his side and not in the

knee shoulder position, and directed to strain down forcibly from time to time.

When the sulcus is not felt the case must be differentiated from volvulus, stricture, internal hernia, pressure on the bowel of outside tumors, and obstruction due to biliary calculi, foreign bodies or impacted feces. These conditions may be readily distinguished by remembering that an acute onset is due to invagination, volvulus or internal hernia. The vomiting remains bilious and not fecal as in perforation and peritonitis, tympanites is less, and the patient voids gas and feces. The temperature is raised in peritonitis and normal in obstruction. Invagination produces partial occlusion, moderate tympanites, bloody stools, tenesmus and palpable tumor. Volvulus may have a history of previous peritonitis or the story of an old hernia that has not come down.

## MUCOCELE OF THE VERMIFORM APPENDIX

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THE following case is of interest because of the comparative rarity of the condition and because of the rather obscure pathological process which it presents.

E.S., American, aged 54, a housewife by occupation, entered the hospital acutely ill, suffering with repeated hæmatamesis. Although ailing for several years her present illness came on suddenly the day before entrance to the hospital. She had felt weak and nauseated for several hours and suddenly vomited a quantity of dark red blood. Two or three hours later she had a second hæmatamesis and this attack left her in such a weakened condition that she was brought to the hospital.

Gastric distress had been present for about three years, but never has been severe enough to require medical attention. She has suffered no real pain; rather more an epigastric discomfort. She states that she always has felt better after eating, but that the distress would reappear one or two hours after the meal. The discomfort would often be relieved by taking small quantities of food, fluids, or baking soda. Occasionally, she would be nauseated, but rarely would vomit. The vomitus never contained any gross blood before the present attack and very seldom any undigested food. These periods of gastric distress would come and go; at intervals she would be entirely free from them. Her appetite has been variable, according to the condition of her general health. She has been failing gradually, however, with a slight loss of weight and an increasing weakness.

Physical examination at the time of entrance revealed the patient almost in extremis from loss of blood. General findings were negative except for the abdominal examination. The abdomen was somewhat retracted, and was lax and flabby. There was only slight tenderness in the upper half, with little or no accompanying rigidity. In the right iliac fossa, however, close to the pelvic brim and seemingly attached to it, was a hard, sausage-shaped mass, which was

exquisitely tender to pressure. It was but slightly movable and seemed to have no pelvic connections. Vaginal examination was negative concerning the pelvic viscera, but high up through the right fornix could be felt the tumor described. It seemed to lie against the wall of the false pelvis and to be attached to it, only slight mobility downward and inward being present. It could be definitely ascertained that there was no connection with the pelvic organs.

A tentative diagnosis of bleeding gastric ulcer and carcinoma of the cæcum was made.

The patient was treated in the usual manner for the hemorrhage, and several days later routine laboratory work was done. Repeated examination of the gastric contents failed to give findings characteristic of either ulcer or malignancy. All röntgenographic work on the stomach was negative for gastric or duodenal pathology. A colon picture, however, with barium clysema, showed a definite filling defect at the cæcum, which was diagnosed as a probable malignancy. As no gastric origin could be found for the hæmatamesis, it was thought that perhaps the cæcal or appendiceal pathology might be the etiological factor. Cases recently have been reported in which profuse gastric hemorrhage has occurred repeatedly, and in which no causative factor could be found other than a chronic appendicitis. Such are thought to be due to a definite toxin causing small erosions of the more superficial arterioles ramifying under the gastric mucosa. All of these cases have completely recovered after removal of the diseased appendix.

The patient had no more hemorrhages and gained strength rapidly, so that she was able to be up and about. Operation was advised and accepted and a laparotomy performed four weeks ago.

Upon opening the peritoneum through a right rectus incision, the mass felt abdominally was exposed and immediately recognized as an enormously dilated appendix, being approximately as large as a man's fist and having an irregular, multi-locular appearance. The walls were thin and distended almost to a solid consistency, and were a glistening white in appearance. The mass lay close against the mesial and inferior surface of the cæcum and was bound to it by fibrinous adhesions. Unfortunately, in freeing it, a portion of the wall was torn and a white, glistening, gelatinous substance was evac-

uated. The lax remnants were easily freed and removed as in the ordinary appendectomy, the stump was inverted and the abdomen was closed without drainage.

The patient has had an uneventful post-operative recovery. The wound has healed by primary union with no tendency to discharge or sinus formation at any time.

Comparatively speaking, such a pathological condition of the appendix is uncommon. Dodge, in reporting his case in 1916, made an exhaustive review of the literature, and was able to collect only 142 cases. A further review since that date has revealed only 18 more reported cases of the condition. So it may be said that such pathology is not of frequent occurrence.

The condition was first described by Virchow in 1863, and since the time of its first recognition has been described by numerous men under a variety of terms. It is of interest to note that a large proportion of the cases reported have been found at autopsy after death from other causes, and that most of those discovered at operation were not suspected before laparotomy.

The condition as recognized has been given a wide terminology: Retention cyst, hydrops, mucocele, colloid cyst, pseudomucinous cyst and cystic disease of the appendix. Perhaps of all these, the two latter describe the chemico-pathological changes most closely. It is a disease entity, inasmuch as any pathological change means disease. It is a cyst in the true definition of the term. Pseudomucinous describes it most exactly chemically, because the tests react positively for that substance and not always for true mucin.

Concerning the etiology of the condition, we have little definite knowledge. It seems to be a true retention cyst, but the change which actually determines the formation of the colloidal material is unknown. Elbe has shown that there are certain underlying conditions which must be present before the process may take place:

*First*, A slowly stenosing process at one or more points of the lumen. Rapid stenosis would mean gangrene.

*Second*, A sterile lumen must be present distal to, or between, points of stenosis. Bacterial invasion, if virulent enough, would mean a secondary empyema of the appendix.

*Third*, There must be an actively secreting mucosa, or at least,

a more rapid secretion than absorption. Added to these factors must be an indefinite change in the mucosa, as yet unknown, whereby the normal secretion is changed into that of a colloidal pseudomucous.

Obliteration of the lumen of the appendix may be brought about by any one of a variety of factors, such as, by a simple chronic inflammation producing fibrosis within the walls, external adhesions, twists or kinks, stenosis of Gerlach's valve, fecal impaction or by the lodgement of foreign bodies. Syphilis and tuberculosis have been given as possible factors and one case has been reported as being caused by an early carcinoma of the appendix wall.

The formation of a cyst is made possible or not by another variety of factors, Bischoff thinks that dilatation is impossible when absorption by the walls takes place in a normal way, *i.e.*, keeps pace with secretion, or when, due to a destruction of the mucous membrane, there no longer can be any secretion into the lumen. Dilatation also does not occur when there is a connection between the cæcum and appendix of sufficient size to allow the secretion to escape into the large bowel. In cases of hydrops (serous accumulation) the stricture of the lumen must be absolute, but with a colloid content, the fluid is of heavy enough consistency to be retained even in the presence of a small canal in the proximal lumen. The above factors must be present, then, before cystic dilatation is possible.

*Pathologically* mucocèles of the appendix present a varied picture. Perhaps the greatest variation is found in the size and shape. The largest specimens reported have approached the size of a man's head, and from such enormous dilatations they have graded downward to sizes hardly larger than a normal appendix.

In shape, the cysts may be sausage, globular, fusiform, egg-shaped, sacculated, single or multi-locular. They may be found occupying any portion of the appendix, or may include the entire organ. The external appearance of the cysts varies with their size, the amount of fluid they contain and the tension which the walls are under. If small, the walls are thick, fibrous appearing and tough. Blood-vessel markings are distinct and there often seems to be an increase in vascularity. As the cyst becomes larger, the walls become thin, translucent, almost of paper thickness, have a dull glistening surface and, due to the pressure, are almost avascular in appearance.

Their consistency also varies with the tension, being at times almost as hard as a solid, fibrous tumor.

The lumen generally is found to be constricted at one or more points by any of the gross changes mentioned above. Changes in the cæcum are rare. Adhesions are also peculiarly seldom found, although a pericæcal veil is sometimes present, binding the cyst and bowel closely together. When adhesions *are* present they generally are fibrinous and are easily separated.

*The contents* of these cysts have been variously described as being mucoid, gelatinous, colloid or pseudomucinous, watery, liquid, serous, or hemorrhagic; a purulent content only is possible in the presence of secondary infection. The color of the cyst content usually is glistening white or yellow. Most frequently the consistency is about that of gelatinous mucus, which is sticky and adheres closely to anything with which it comes in contact. In the heaviest type, it is almost the consistency of jelly and seems to be held together by fine fibrinous tendrils. With this exception, microscopically the substance has no structure. Chemically, the contents give a positive test for pseudomucin; rarely a reaction for true mucin is given.

Those cases in which the cyst content is serous and not of a mucoid character, cannot truly be designated as "mucocèle," but are rather hydropic conditions. This latter is a much rarer condition; there is a lessened production of mucus, and complete atresia of the lumen at some proximal point is necessary to hold the serous content. Clinically, however, they are classified the same as the "mucocèle."

*Histologically* the appendix shows changes of a chronic inflammation in those areas not occupied by the cyst *per se*; thickening, round celled infiltration or fibrosis, entirely dependent upon the acuteness or duration of the inflammatory change. At the site of the cyst the condition varies with the size; if small, the muscularis and mucosa are thickened, the goblet cells are full and active and there is passive hyperæmia present. As the cyst enlarges, resulting pressure changes take place. The mucosa, muscularis and serosa become thinned, and if sufficient tension occurs, gradually undergo a pressure atrophy and the original structure is replaced by fibrous tissue. The lymph follicles and glandular elements also undergo

a flattening along with this change, and gradually disappear, to be replaced by fibrous elements. Histologically there are no findings to indicate a malignant character of these cysts, but clinically they are classed as being potentially malignant. This term, however, must be used in a broad sense, because they do not fill all the pathological requirements generally considered of a malignant growth. Analogous to the papillary cystadenoma of the ovary, a rupture of the cyst is generally followed by a reactionary peritonitis of a progressive character. This condition has been termed pseudomyxomatous peritonitis. Seelig, in his recent article, states that it is caused in the male only by a rupture of a mucocele of the appendix. The exact mode of production of this type of peritonitis has not definitely been established. Some hold that it is a reaction merely to mechanical irritation by the escaped colloid acting on peritoneal surfaces. This, however, would not account for the large amounts of pseudomucin found free in the abdominal cavity in such a case. With this in view, others hold that a myxomatous degeneration of the peritoneum must take place as a result of this irritation, with a consequent formation of more of the mucoid material. Still others believe that escaped specialized epithelial cells implant themselves on the peritoneal surfaces and go on producing their secretion without control. Which one, or combination, of these theories is correct cannot be stated at the present time. We know, however, that the affair spreads both by continuity and contiguity and that invasion of parenchymatous organs, such as the liver and spleen, does take place. The condition has never been known to metastasize either by the lymphatics or blood stream. It is further peculiar in its malignant tendency, inasmuch as the substance, as long as it is retained within its cyst wall, is benign.

*The symptoms* caused by these pseudomyxomatous cysts are characteristically vague and indefinite. In many cases reported there has been a chronic digestive syndrome, which might have been due either to stomach, gall-bladder or appendix. Others have had chronic symptoms referable to the right iliac fossa. In women, symptoms many times are those of menstrual disturbances, or subjective feelings more or less characteristic of pelvic pathology. Bladder irritability has been noted rather commonly, especially in women. Many cases

reported have given no direct symptoms at all, but the condition has been found at operation on other viscera. And finally there is that large proportion of cases which have been quiescent, and have been found only at autopsy after death from some other cause. The diagnosis rarely is made; many times appendiceal pathology is known to be present, but the true condition is very seldom recognized pre-operatively.

*The treatment*, of course, is appendectomy. Those cases reported as operated have uniformly gone on to an uneventful recovery, unless one of the numerous surgical complications caused a fatality. But the potential malignancy of the condition must be remembered and all care used that none of the cyst content escapes to soil the peritoneum. Even a small amount, if a generalized peritonitis does not result, may be the cause of a chronic sinus, discharging the mucinous material. Although this patient so far has had an uneventful post-operative recovery, she must be observed at frequent intervals over a long period of time, before it can be said that no peritoneal sequelæ have occurred.

#### SUMMARY

1. Mucocèles of the vermiform appendix are comparatively rare conditions. To date approximately 160 cases have been reported.

2. They are true retention cysts, probably caused by chronic inflammatory changes.

3. They are potentially malignant. The metastases, however, do not occur until after rupture of the cyst and the pseudomucin has come in contact with the peritoneum. They are the only cause of pseudomyxomatous peritonitis in the male and one of the two causes in the female.

4. The trend of symptoms caused by the condition is vague and indefinite and such that a positive diagnosis rarely is possible.

5. In the differential diagnosis of tumors in the right iliac region, mucocele of the appendix always should be considered.



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(THIRTY-SECOND SERIES)

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